09/095683

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FILE 'USPAT' ENTERED AT 16:04:29 ON 15 DEC 1998

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  *                W E L C O M E   T O   T H E                    *
  *          U. S.   P A T E N T   T E X T   F I L E              *
  * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * * *

=> s flow channel

        818295 FLOW
        336806 CHANNEL
L1        7844 FLOW CHANNEL
               (FLOW(W)CHANNEL)

=> s  optical

L2      278405 OPTICAL

=> s l1 and l2

L3         705 L1 AND L2

=> s reading

L4      263218 READING

=> s l3 and l4

L5         142 L3 AND L4

=> s vent

L6       66614 VENT

=> s l5 and l6

L7          22 L5 AND L6

=> s kinase or dehydrogenase

          8719 KINASE
          6666 DEHYDROGENASE
L8       13169 KINASE OR DEHYDROGENASE

=> s l7 and l8

L9           1 L7 AND L8

=> d l9


1.  5,059,654, Oct. 22, 1991, Affinity matrices of modified
polysaccharide supports; Kenneth C. Hou, et al., 525/54.1; 210/198.2,
502.1, 656; 422/59, 70, 89; 435/180; 525/54.2, 54.21; 530/391.1, 391.5,
413; 536/23.1 [IMAGE AVAILABLE]

=> s diaphorase
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09/095683

L10 386 DIAPHORASE

=> s l7 and l10

L11 0 L7 AND L10

09/095683

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=> s channel (p) layer

        338878 CHANNEL
        539690 LAYER
L1       30372 CHANNEL (P) LAYER

=> s optical

L2      280534 OPTICAL

=> s l1 and l2

L3        5239 L1 AND L2

=> s kinase or dehydrogenase

          9056 KINASE
          6811 DEHYDROGENASE
L4       13569 KINASE OR DEHYDROGENASE

=> s l3 and l4

L5          27 L3 AND L4

=> d l5 1-27
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1. 5,801,057, Sep. 1, 1998, Microsampling device and method of construction; Wilson H. Smart, et al., 436/68, 95; 600/309 [IMAGE AVAILABLE]

2. 5,798,380, Aug. 25, 1998, Cytoskeletal active agents for glaucoma therapy; Paul L. Kaufman, et al., 514/456, 913 [IMAGE AVAILABLE]

3. 5,789,197, Aug. 4, 1998, Microsomal triglyceride transfer protein; John R. Wetterau, II, et al., 435/69.1, 15, 320.1; 530/350; 536/23.5 [IMAGE AVAILABLE]

4. 5,770,688, Jun. 23, 1998, Method and composition for the treatment of mammalian HIV infection; Torben Saermark, et al., 530/324 [IMAGE AVAILABLE]

5. 5,747,274, May 5, 1998, Method and device for diagnosing and distinguishing chest pain in early onset thereof; George Jackowski, 435/7.94; 422/56, 58, 60, 61; 435/7.93, 970, 973, 975; 436/161, 164, 514, 528, 530, 531, 807, 808, 810, 811 [IMAGE AVAILABLE]

6. 5,744,358, Apr. 28, 1998, Method and device for diagnosing and distinguishing chest pain in early onset thereof; George Jackowski, 435/7.4; 422/56, 58, 60, 61; 435/7.94, 970, 973, 975; 436/161, 164, 514, 528, 530, 531, 807, 808, 810, 811 [IMAGE AVAILABLE]

7. 5,723,345, Mar. 3, 1998, Method and device for specific binding assay; Tadakazu Yamauchi, et al., 436/518; 204/400, 403; 422/57, 58, 82.01, 82.05, 82.08, 82.09; 435/7.1, 7.72, 7.9, 7.91, 7.92, 287.1, 287.2, 817; 436/169, 514, 531, 535, 536, 538, 805, 806 [IMAGE AVAILABLE]

8. 5,710,008, Jan. 20, 1998, Method and device for diagnosing and

distinguishing chest pain in early onset thereof; George Jackowski, 435/7.4; 422/56, 58; 435/7.94, 970, 973, 975; 436/514, 528, 530, 807, 808, 810 [IMAGE AVAILABLE]

9. 5,643,721, Jul. 1, 1997, Bioreagent immobilization medium; Thomas G. Spring, et al., 435/6; 204/290R, 400, 403, 409; 252/500, 502, 503, 504, 506, 507, 508, 510, 511, 514, 516, 519.32, 519.33; 422/82.01; 435/178, 179, 180, 817; 436/518, 524, 528, 806 [IMAGE AVAILABLE]

10. 5,604,105, Feb. 18, 1997, Method and device for diagnosing and distinguishing chest pain in early onset thereof; George Jackowski, 435/7.4; 422/56, 58; 435/7.94, 970, 973, 975; 436/514, 528, 530, 807, 808, 810 [IMAGE AVAILABLE]

11. 5,595,872, Jan. 21, 1997, Nucleic acids encoding microsomal trigyceride transfer protein; John R. Wetterau, II, et al., 435/6, 320.1; 536/23.1 [IMAGE AVAILABLE]

12. 5,520,787, May 28, 1996, Diagnostic flow cell device; Ted J. Hanagan, et al., 204/409, 403, 412, 415; 205/777.5; 422/68.1, 82.01, 82.02; 435/287.1, 287.7, 287.9, 817 [IMAGE AVAILABLE]

13. 5,457,053, Oct. 10, 1995, Reagent container for analytical rotor; Tammy L. Burd, et al., 436/45; 422/72 [IMAGE AVAILABLE]

14. 5,304,348, Apr. 19, 1994, Reagent container for analytical rotor; Tammy L. Burd, et al., 422/72; 436/45; 494/45 [IMAGE AVAILABLE]

15. 5,242,606, Sep. 7, 1993, Sample metering port for analytical rotor having overflow chamber; Boris Braynin, et al., 210/787, 380.1; 422/72, 119, 918, 947; 436/45, 180; 494/17 [IMAGE AVAILABLE]

16. 5,186,844, Feb. 16, 1993, Apparatus and method for continuous centrifugal blood cell separation; Tammy L. Burd, et al., 210/782, 95, 198.1, 380.1, 514, 532.1, 789; 422/72, 101; 436/45, 63, 177, 180; 494/27, 34, 43 [IMAGE AVAILABLE]

17. 5,173,193, Dec. 22, 1992, Centrifugal rotor having flow partition; Carol T. Schembri, 210/782, 95, 198.1, 380.1, 513, 745; 422/64, 72, 101, 102; 436/45, 63, 177, 180 [IMAGE AVAILABLE]

18. 5,122,284, Jun. 16, 1992, Apparatus and method for optically analyzing biological fluids; Boris Braynin, et al., 210/782, 94, 198.1, 380.1, 514, 532.1, 745, 789; 356/246, 427; 422/72, 102; 436/45, 63, 177, 180; 494/10, 17, 29, 37, 43 [IMAGE AVAILABLE]

19. 5,061,381, Oct. 29, 1991, Apparatus and method for separating cells from biological fluids; Tammy L. Burd, 210/789, 94, 198.1, 380.1, 514, 515, 532.1; 422/64, 72, 101, 102; 436/45, 63, 177, 180; 494/16, 17, 29, 37, 43 [IMAGE AVAILABLE]

20. 5,059,654, Oct. 22, 1991, Affinity matrices of modified polysaccharide supports; Kenneth C. Hou, et al., 525/54.1; 210/198.2, 502.1, 656; 422/59, 70, 89; 435/180; 525/54.2, 54.21; 530/391.1, 391.5, 412, 413; 536/23.1 [IMAGE AVAILABLE]

21. 4,908,112, Mar. 13, 1990, Silicon semiconductor wafer for analyzing micronic biological samples; Salvatore J. Pace, 210/198.2; 204/601, 612, 616; 210/251; 250/461.2; 356/318, 344 [IMAGE AVAILABLE]

22. 4,761,381, Aug. 2, 1988, Volume metering capillary gap device for applying a liquid sample onto a reactive surface; Joel M. Blatt, et al., 436/165; 356/246; 422/57, 58, 102, 947; 436/166 [IMAGE AVAILABLE]

23. 4,610,544, Sep. 9, 1986, Flow analysis; Clifford Riley, 356/410;

422/64, 82; 436/53 [IMAGE AVAILABLE]

24. 4,486,097, Dec. 4, 1984, Flow analysis; Clifford Riley, 356/410; 422/64, 82 [IMAGE AVAILABLE]

25. 4,264,560, Apr. 28, 1981, Clinical analytical system; Samuel Natelson, 422/58; 356/246; 422/57, 61, 66, 81 [IMAGE AVAILABLE]

26. 4,259,079, Mar. 31, 1981, Method and apparatus for electrical separation of molecules; Alvin S. Blum, 204/518, 627; 422/81, 82; 435/283.1, 287.1 [IMAGE AVAILABLE]

27. RE 29,725, Aug. 8, 1978, Analytical test pack and process for analysis; Donald R. Johnson, et al., 435/12; 206/219; 356/246, 409; 422/61, 915, 940; 435/14, 16, 287.6, 288.5, 810; 436/95, 108, 165 [IMAGE AVAILABLE]

=> s vent

L6 66926 VENT

=> s l5 and l6

L7 6 L5 AND L6

=> d l7 1-6

1. 5,801,057, Sep. 1, 1998, Microsampling device and method of construction; Wilson H. Smart, et al., 436/68, 95; 600/309 [IMAGE AVAILABLE]

2. 5,242,606, Sep. 7, 1993, Sample metering port for analytical rotor having overflow chamber; Boris Braynin, et al., 210/787, 380.1; 422/72, 119, 918, 947; 436/45, 180; 494/17 [IMAGE AVAILABLE]

3. 5,186,844, Feb. 16, 1993, Apparatus and method for continuous centrifugal blood cell separation; Tammy L. Burd, et al., 210/782, 95, 198.1, 380.1, 514, 532.1, 789; 422/72, 101; 436/45, 63, 177, 180; 494/27, 34, 43 [IMAGE AVAILABLE]

4. 5,061,381, Oct. 29, 1991, Apparatus and method for separating cells from biological fluids; Tammy L. Burd, 210/789, 94, 198.1, 380.1, 514, 515, 532.1; 422/64, 72, 101, 102; 436/45, 63, 177, 180; 494/16, 17, 29, 37, 43 [IMAGE AVAILABLE]

5. 5,059,654, Oct. 22, 1991, Affinity matrices of modified polysaccharide supports; Kenneth C. Hou, et al., 525/54.1; 210/198.2, 502.1, 656; 422/59, 70, 89; 435/180; 525/54.2, 54.21; 530/391.1, 391.5, 412, 413; 536/23.1 [IMAGE AVAILABLE]

6. 4,761,381, Aug. 2, 1988, Volume metering capillary gap device for applying a liquid sample onto a reactive surface; Joel M. Blatt, et al., 436/165; 356/246; 422/57, 58, 102, 947; 436/166 [IMAGE AVAILABLE]

=> s diaphorase

L8 389 DIAPHORASE

=> s l5 and l8

L9 3 L5 AND L8

=> d l9 1-3

1. 5,801,057, Sep. 1, 1998, Microsampling device and method of construction; Wilson H. Smart, et al., 436/68, 95; 600/309 [IMAGE AVAILABLE]

2. 5,723,345, Mar. 3, 1998, Method and device for specific binding assay; Tadakazu Yamauchi, et al., 436/518; 204/400, 403; 422/57, 58, 82.01, 82.05, 82.08, 82.09; 435/7.1, 7.72, 7.9, 7.91, 7.92, 287.1, 287.2, 817; 436/169, 514, 531, 535, 536, 538, 805, 806 [IMAGE AVAILABLE]

3. 4,761,381, Aug. 2, 1988, Volume metering capillary gap device for applying a liquid sample onto a reactive surface; Joel M. Blatt, et al.,

Leary
09/095683

=> fil reg; d que l1; d que l2; fil caplu; d que l6

FILE 'REGISTRY' ENTERED AT 14:53:52 ON 26 OCT 1998
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

STRUCTURE FILE UPDATES: 23 OCT 98 HIGHEST RN 213175-60-1
DICTIONARY FILE UPDATES: 25 OCT 98 HIGHEST RN 213175-60-1

TSCA INFORMATION NOW CURRENT THROUGH JUNE 29, 1998

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Stereochemical name changes have been adopted and appear in CN's beginning 6/29/98. See the online news message for details.

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- key terms

L1 10 SEA FILE=REGISTRY ABB=ON PLU=ON (HEXOKINASE OR GLUCOSE DEHYDROGENASE OR DIAPHORASE)/CN

L2 2 SEA FILE=REGISTRY ABB=ON PLU=ON GLUCOSE/CN

FILE 'CAPLUS' ENTERED AT 14:53:53 ON 26 OCT 1998
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1967 - 26 Oct 1998 VOL 129 ISS 18
FILE LAST UPDATED: 26 Oct 1998 (981026/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file supports REG1stRY for direct browsing and searching of

all substance data from the REGISTRY file. Enter HELP FIRST for more information.

```
L1          10 SEA FILE=REGISTRY ABB=ON  PLU=ON  (HEXOKINASE OR GLUCOSE
               DEHYDROGENASE OR DIAPHORASE)/CN
L2           2 SEA FILE=REGISTRY ABB=ON  PLU=ON  GLUCOSE/CN
L3       17014 SEA FILE=CAPLUS ABB=ON  PLU=ON  L1 OR HEXOKINASE OR HEXO
               KINASE OR GLUCOSE(W)(DEHYDROGENASE OR DE HYDROGENASE) OR
               DIAPHORASE
L4        7762 SEA FILE=CAPLUS ABB=ON  PLU=ON  L3 AND (L2 OR GLUCOSE OR
               ANALYTE)
L5        1782 SEA FILE=CAPLUS ABB=ON  PLU=ON  L4 AND (BLOOD OR PLASMA
               OR FLUID OR SPECIMEN OR SAMPL?)
L6          22 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 AND (CHAMBER? OR
               COMPART?)

=> d 1-22 .bevstr


L6   ANSWER 1 OF 22  CAPLUS  COPYRIGHT 1998 ACS
AN   1998:585340  CAPLUS
DN   129:172753
TI   Microsampling device and method of construction
IN   Smart, Wilson H.; Subramanian, Kumar
PA   USA
SO   U.S., 10 pp.
     CODEN: USXXAM
PI   US 5801057 A   19980901
AI   US 96-620994  19960322
DT   Patent
LA   English
AB   A minimally intrusive and less painful, self-use microsampling
     device and method for the measurement of glucose and other
     analytes in blood are provided.  The device of the
     invention may have one or two optical windows for measuring the
     concn. of an absorbent reaction product or no windows if methods
     other than optical absorbance is used.  The sampling
     chamber of the device can contain anal. reagents and other
     additives to facilitate the sampling and anal. steps.
     Also provided is a fabrication method for the microsampling device.
IT   50-99-7, D-Glucose, analysis
     RL: ANT (Analyte); ANST (Analytical study)
        (microsampling device and method of construction)
IT   9028-53-9, Glucose dehydrogenase
     RL: ARG (Analytical reagent use); DEV (Device component use); ANST
     (Analytical study); USES (Uses)
        (microsampling device and method of construction)
```

L6 ANSWER 2 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1998:528771 CAPLUS
TI Improved accuracy biosensor strip for accuchek advantage
AU Surridge, N. A.; Burke, D.; Diebold, E.; Delk, R. D.
CS Boehringer Mannheim Corp., Indianapolis, IN, 46250, USA
SO Book of Abstracts, 216th ACS National Meeting, Boston, August 23-27 (1998), I&EC-062 Publisher: American Chemical Society, Washington, D. C.
CODEN: 66KYA2
DT Conference; Meeting Abstract
LA English
AB A second generation biosensor test strip has been developed for the highly successful AccuChek Advantage **blood glucose** monitoring system. The measurement methodol. of the new strip is the same as that of the strip currently sold to the hospital market in the US, and uses the enzyme **Glucose Dehydrogenase** in combination with a biamperometric measurement to det. **glucose** levels in capillary, venous, arterial and neonatal **blood samples**. However, the biosensor described here employs a capillary-fill design to reduce the **blood** vol. required to 4 .mu.L. In addn., the reagent on the test strip has been specifically developed to control the dynamic properties of the reagent film as the **sample** enters the capillary **chamber** and dissoln. occurs. Through the addn. of a specific polymer, it is possible to impart unique properties to the reagent which cause the developing diffusion layer during the measurement to have a high degree of uniformity from strip-to-strip. This results in excellent Total System Error of between 12 and 16%, reduced sensitivity of the reagent to the hematocrit level of the individual **sample**, as well as reduced sensitivity to environmental effects, all leading to enhanced accuracy.

L6 ANSWER 3 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1998:224846 CAPLUS
DN 128:267777
TI **Glucose** metabolism in human malignant gliomas measured quantitatively with PET, 1-[C-11]**glucose** and FDG: analysis of the FDG lumped constant
AU Spence, Alexander M.; Muzi, Mark; Graham, Michael M.; O'sullivan, Finbarr; Krohn, Kenneth A.; Link, Jeanne M.; Lewellen, Thomas K.; Lewellen, Barbara; Freeman, Scott D.; Berger, Mitchel S.; Ojemann, George A.
CS Departments of Neurology, Radiology, Statistics and Neurological Surgery, University of Washington School of Medicine, Seattle, WA, 98195, USA
SO J. Nucl. Med. (1998), 39(3), 440-448
CODEN: JNMEAQ; ISSN: 0161-5505
PB Society of Nuclear Medicine

DT Journal
LA English
AB Calcn. of the **glucose** metabolic rate (MRGlc) in brain with PET and 2-[18F]fluoro-2-deoxy-D-**glucose** (FDG) requires knowing the rate of uptake of FDG relative to **glucose** from **plasma** into metabolite pools in the tissue. The proportionality factor for this is the FDG lumped const. (LCFDG), the ratio of the vols. of distribution of FDG and **glucose** multiplied by the **hexokinase** phosphorylation ratio for the two hexoses, KmGlcVmrdgFDG/KmFDG.cntdot.VMGlc. MRGlc equals the FDG metabolic rate (MRFDG) divided by the LCFDG, i.e., MRGlc = MRFDG/LCFDG and LCFDG = MRFDG/MRGlc. This investigation tested the hypothesis that LCFDG is significantly higher in gliomas than it is in brain uninvolved with tumor. We imaged 40 patients with malignant gliomas with 1-[11C]**glucose** followed by FDG. The metabolic rates MRGlc and MRFDG were estd. for glioma and contralateral brain regions of interest by an optimization program based on three-**compartment**, four-rate const. models for the two hexoses. The LCFDG, estd. as MRFDG/MRGlc, in gliomas was 1.40 .+-. 0.46 (mean .+-. s.d.; range = 0.72-3.10), whereas in non-tumor-bearing contralateral brain, it was 0.86 .+-. 0.14 (range = 0.61-1.21) ($p < 0.001$, glioma vs. contralateral brain). These data strongly suggest that the glioma LCFDG exceeds that of contralateral brain, that quantitation of the glioma MRGlc with FDG requires knowing the LCFDG specific for the glioma and that the LCFDG of normal brain is higher than previously reported ests. of about 0.50. 2-Fluoro-2-deoxy-D-**glucose**/PET studies in which glioma **glucose** metab. is calcd. by the autoradiog. approach with normal brain rate consts. and LCFDG will overestimate glioma MRGlc, to the extent that the glioma LCFDG exceeds the normal brain LCFDG. "Hot spots" visualized in FDG/PET studies of gliomas represent regions where MRGlc, LCFDG or their product is higher in glioma than it is in uninvolved brain tissue.
IT **50-99-7**, **Glucose**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**glucose** metab. in human malignant gliomas measured quant. with PET, 1-[C-11]**glucose** and FDG)

L6 ANSWER 4 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1997:513584 CAPLUS
DN 127:146829
TI Indicator systems and material compression and insertion devices for preparing same
IN Hendricks, Judy K.; Biddle, Harold A.; Byerly, Dale L.; Rechsteiner, Shaundrea L.; Gorski, Joel R.
PA North American Science Associates, Inc., USA
SO PCT Int. Appl., 79 pp.
CODEN: PIXXD2

PI WO 9726924 A1 19970731
DS W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 97-US554 19970122
PRAI US 96-10312 19960122
US 96-25514 19960905
US 96-735992 19961024
US 96-736310 19961024
DT Patent
LA English
AB This invention relates to novel app. and methods for inserting and positioning a compressible material into a container and for using the container for detecting a specific environmental parameter or combination of parameters and for detg. the effectiveness of a sterilization procedure. Precise positioning of a plug of compressible material in a container provides flexibility for prodn. of indicator systems that vary in their response to sterilizing conditions. These indicators reflect the efficacy of sterilizers based on different modes of sterilization and reproducibility for accurate monitoring of each mode. The invention also relates to test indicators contg. controlled vols. of compressed, gas-permeable materials and to methods for using test indicators for detg. the efficacy of different types of sterilization processes. The test indicator consists of a plurality of interactive enzymes in a container with at least one opening. The opening is filled with a compressed cylindrical foam insert and the test indicator is placed into the sterilization **chamber**. The foam insert regulates the amt. of sterilant such as steam, gas, chems. or **plasma** entering the test indicator. Upon proper sterilization, the sterilant destroys the interactive enzymes and no color product is formed. Inactivation of the enzyme system parallels the inactivation of bacterial spores subjected to the sterilization process. Results are available in from a few seconds to a few hours. The test indicator can also be placed into a container with material such that the design simulates an environmental parameter test of the sterilization process.
IT **9028-53-9, Glucose dehydrogenase**
37340-89-9, Diaphorase
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(indicator systems and material compression and insertion devices for prepg. same)

L6 ANSWER 5 OF 22 CAPLUS COPYRIGHT 1998 ACS
Searcher : Shears 308-4994

AN 1997:79847 CAPLUS
DN 126:168597
TI Effects of pressure on whole **blood glucose** measurements using the Bayer Glucometer 4 **blood glucose** meter
AU Edge, C.J.; Grieve, A. P.; Gibbins, N.; O'sullivan, F.; Bryson, P.
CS Diving Diseases Research Centre, Plymouth, PL6 8BQ, UK
SO Undersea Hyperbaric Med. (1996), 23(4), 221-224
CODEN: UHMEE7; ISSN: 1066-2936
PB Undersea and Hyperbaric Medical Society
DT Journal
LA English
AB Effects of pressure on whole **blood glucose** measurements using the Bayer Glucometer 4 **blood glucose** meter. Undersea Hyperbaric Med 1996; 23(4):221-224.-The effect of pressure was investigated on the readings of whole **blood glucose** obtained from the Bayer Glucometer 4 **blood glucose** meter which uses the **hexokinase** enzymic reaction. Sixteen subjects (eight normal and eight insulin-dependent diabetics) were exercised in a hyperbaric **chamber** at a depth of 3.7 atm abs. Venous **blood samples** were monitored at regular intervals for whole **blood glucose** concn. as measured by a Glucometer 4 inside the **chamber**. The **blood samples** were immediately placed in an airlock and taken to 1 atm abs, where whole **blood glucose** concns. were measured using an identical instrument. The remaining **blood** was then analyzed in duplicate for serum **glucose** concn. using std. lab. methods. The results show a significant difference between whole **blood glucose** concns. measured at pressure and those measured at atm. pressure. Significant differences are also obsd. between whole **blood glucose** concns. measured under pressure and serum **blood glucose** concns. measured at atm. pressure.
IT **50-99-7**, **Glucose**, analysis
RL: ANT (Analyte); ANST (Analytical study)
(effects of pressure on whole **blood glucose** measurements using the Bayer Glucometer 4 **blood glucose** meter)

L6 ANSWER 6 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1996:490052 CAPLUS
DN 125:164825
TI The metabolic organization of the adult human liver: A comparative study of normal, fibrotic, and cirrhotic liver tissue
AU Racine-Samson, Lorraine; Scoazec, Jean-Yves; D'Errico, Antonia; Fiorentino, Michelangelo; Christa, Laurence; Moreau, Alain; Roda, Corrado; Grigioni, Walter F.; Feldmann, Gerard
CS Faculte de Medecine Xavier Bichat, Universite Denis Diderot, Paris,

75870/18, Fr.
SO Hepatology (Philadelphia) (1996), 24(1), 104-113
CODEN: HPTLD9; ISSN: 0270-9139
DT Journal
LA English
AB Little is known about the alterations of metabolic organization of the human liver tissue in chronic liver diseases. The authors therefore compared the distribution of the following zonal metabolic markers in 10 **samples** of normal liver tissue, 10 **samples** of fibrotic tissue, and 22 **samples** of cirrhotic tissue: (a) the enzymic activities of **glucose** 6-phosphatase (G6P), lactate dehydrogenase (LDH), succinate dehydrogenase (SDH), nicotinamide-adeninedinucleotide-phosphate [NADPH] dehydrogenase (ND), .beta.-hydroxybutyrate dehydrogenase (HBDH), and glutamate dehydrogenase (GDH); (b) the protein glutamine synthetase (GLS); and (c) albumin mRNA. The normal human hepatic lobule was characterized by the periportal predominance of G6P and SDH enzymic activities and albumin mRNAs, the perivenous predominance of ND and GDH, the restriction of GLS to a small perivenous **compartment**, and the predominance of .beta.-HBDH at the contact of both portal tracts and centrilobular veins. In fibrosis, the overall metabolic organization of the normal liver tissue was retained. The expression of periportal markers predominated around enlarged portal tracts and that of perivenous markers around residual centrilobular veins. GLS was constantly detected at the contact of centrilobular veins. In cirrhotic nodules, no zonation was obsd. for most enzymic activities or for albumin. Only G6P usually predominated at the periphery of the nodules. GLS was constantly undetectable. No difference according to the etiol. of the underlying disease was obsd. In conclusion, the normal human hepatic lobule presents a marked metabolic zonation, preserved in fibrotic lesions, but lost in cirrhotic nodules. The alterations of the metabolic organization obsd. in cirrhosis might contribute to the pathogenesis of some of the metabolic disorders assocd. with advanced liver disease.
IT **9001-68-7**, NADPH dehydrogenase
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(metabolic organization of human liver tissue in fibrosis and cirrhosis in comparison with normal liver tissue)

L6 ANSWER 7 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1996:76827 CAPLUS
DN 124:198703
TI **Compartment** analysis of cerebral **glucose** metabolism and in vitro **glucose**-metabolizing enzyme activities in the rat brain
AU Ouchi, Yasuomi; Fukuyama, Hidenao; Matsuzaki, Shigeru; Ogawa, Masafumi; Kimura, Jun; Tsukada, Hideo; Kakiuchi, Takeharu; Kosugi,

Tsuyoshi; Nishiyama, Shingo
CS Department of Neurology, Faculty of Medicine, Kyoto University, 54 Shogoin Kawahara-cho, Sakyo-ku, Kyoto, Japan
SO Brain Res. (1996), 706(2), 267-72
CODEN: BRREAP; ISSN: 0006-8993
DT Journal
LA English
AB To clarify the relation between cerebral **glucose** metabolic rate consts. and **glucose**-metabolizing enzyme activities in the cerebral cortex, the authors evaluated the cerebral metabolic rate of **glucose** (CMRGlu), metabolic rate consts. of [18F]-2-fluoro-2-deoxy-D-**glucose** (FDG) and related enzyme activities in the frontal cortex under normal and **glucose** metab.-suppressed conditions. Applying a three-**compartment** four-parameter model, metabolic rate consts. were obtained by dynamic positron emission tomog. with FDG, and CMRGlu was calcd. based on these rate consts. The glycolytic enzyme activities were detd. by in vitro biochem. assay. Three days after ibotenic acid injection into the basal forebrain, CMRGlu was decreased in the ibotenic acid-treated frontal cortex as well as k3* (phosphorylation), while K1* (**plasma** to brain) showed no remarkable change. No significant redns. of the enzyme activities except for **hexokinase** activity were found in the frontal cortex. Regression anal. showed a significant pos. correlation between k3* and the **hexokinase** activity. These results suggested that k3* in the **compartment** anal. reflects **hexokinase** activity.
IT **9001-51-8**, **Hexokinase**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(**compartment** anal. of cerebral **glucose** metab. and in vitro **glucose**-metabolizing enzyme activities in rat brain)
IT **50-99-7**, D-**Glucose**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**compartment** anal. of cerebral **glucose** metab. and in vitro **glucose**-metabolizing enzyme activities in rat brain)

L6 ANSWER 8 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1995:922953 CAPLUS
DN 124:51634
TI Net sugar transport is a multistep process. Evidence for cytosolic sugar binding sites in erythrocytes
AU Cloherty, Erin K.; Sultzman, Lisa A.; Zottola, Ralph J.; Carruthers, Anthony
CS Medical School, University of Massachusetts, Worcester, MA, 01605, USA

SO Biochemistry (1995), 34(47), 15395-406
CODEN: BICHAW; ISSN: 0006-2960
DT Journal
LA English
OS CJACS

AB Human erythrocyte net sugar transport is hypothesized to be rate-limited by reduced cytosolic diffusion of sugars and/or by reversible sugar assocn. with intracellular macromols. (Naftalin, R. J. et al., 1985). The present study examines these hypotheses. Protein-mediated 3-O-methylglucose uptake at 4.degree. by human erythrocytes and by resealed, hypotonically lysed erythrocytes (ghosts) is inhibited by increasing solvent viscosity. Protein-mediated transport and transbilayer diffusion of the slowly transported substrate 6-nitrobenzoxadiazolyl glucosamine are unaffected by increasing solvent viscosity. These findings suggest that protein-mediated 3-O-methylglucose transport is diffusion-limited in erythrocytes. More detailed analyses of red cell 3-O-methylglucose uptake (at 4.degree. and at limiting extracellular sugar levels) reveal that net influx is a biexponential process characterized by rapid filling of a small **compartment** (C1 = 29% total cell vol.; k1 = 7.4 min-1) and slow filling of a larger **compartment** (C2 = 71% total cell vol.; k2 = 0.56 min-1). Erythrocyte D-**glucose** net uptake at 4.degree. is also a biphasic process. Transmembrane sugar leakage is a monoexponential process indicating that multicomponent, protein-mediated uptake does not result from sugar uptake by two cell populations of differing cellular vol. Sugar exit at limiting 3-O-methylglucose concns. is described by single exponential kinetics. This demonstrates that multicomponent sugar uptake does not result from influx into two populations of cells with widely different sugar transporter content. The authors conclude that biexponential sugar uptake results from slow (relative to transport) exchange of sugars between serial, intracellular sugar **compartments**. Biexponential sugar uptake is obsd. under equil. exchange conditions (intracellular sugar concn. = extracellular sugar concn.) but only at 3-O-methylglucose concns. of less than 1 mM. Above this sugar concn., exchange uptake is a monoexponential process. Because diffusion rates are independent of diffusant concn., this suggests that multicomponent uptake results from high-affinity sugar binding within the cell. The concn. of cytosolic binding sites (30 .mu.M, Kd(app) = 400 .mu.M) was estd. from the equil. cellular 3-O-methylglucose space. Biexponential net 3-O-methylglucose uptake is also obsd. in human erythrocyte ghosts, in control human K562 cells, and in K562 cells induced to synthesize Hb by prolonged exposure to hemin. This demonstrates that neither membrane-bound nor free cytosolic Hb forms the sugar-binding complex. .alpha.-Toxin-permeabilized cells fill rapidly (within 5 s) with 3-O-methylglucose and L-**glucose** (a nontransported sugar), indicating that the **glucose** binding

compartment does not extend across the entire intracellular margin of the **plasma** membrane. Rather, it must be restricted to domains of locally high-**glucose** transporter d. Immunofluorescence microscopy of erythrocytes indicates that GLUT1 is not distributed uniformly across the cell surface, while the anion transporter shows a uniform cell surface distribution. Red cell **hexokinase** I and GLUT1 appear not to colocalize in hypotonically lysed erythrocytes. The kinetics of sugar uptake and exit are quant. mimicked by a model in which newly imported sugars enter the bulk intracellular water only following interaction with an intracellular, sugar-binding complex. The authors conclude that steady state sugar transport assays in human erythrocytes measure two processes: rapid sugar translocation across the bilayer and slow sugar release into bulk cytosol. The conclusions of previous steady state analyses which assume net transport reflects only sugar translocation may require reconsideration.

IT **50-99-7**, D-**Glucose**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(multistep processes and cytosolic sugar-binding sites in sugar transport by human erythrocytes)

L6 ANSWER 9 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1994:574431 CAPLUS
DN 121:174431
TI Cerebral circulation and metabolism measurement using positron emission tomography
AU Kanno, Iwao
CS Department Radiology and Nuclear Medicine, Akita Research Institute Brain and Blood Vessels, Akita, 010, Japan
SO Shinkei Kenkyu no Shinpo (1994), 38(2), 256-64
CODEN: SKNSAF; ISSN: 0001-8724
DT Journal
LA Japanese
AB Oxygen extn. fraction (OEF) was measured with 15O2 inhalation. Because of a short half-life, 15O tracer has an advantage to apply the steady-state approach for cerebral **blood** flow and oxygen metab. (CMRO2), and the other advantage is repeatability of the study. The repeatability is an important feature to evaluate pathol. brain functions like circulation reserve, metabolic reserve, and responsibility to perturbations by specific drug effect. The repeatability is also strong advantage as a tool for the activation study. **Glucose** metab. can be measured by 18F labeled fluorodeoxyglucose (FDG). The FDG transports **blood** brain barrier (BBB) like natural **glucose** and being phospholyzed by the **hexokinase**, but not being further metabolized unlike the **glucose**. Since the phospholyzed FDG will stay at the site, the **glucose** consumption can be calcd. based on this concn., the lumped const. which is detd. by competition of

the natural **glucose** and FDG, and kinetic rate consts. which are detd. through three-**compartment** model. It is worthwhile to know in interpretation of PET measurement, that FDG method will provide the entrance of the energy metab. though the oxygen metab. will provide the later stage of it. Simultaneous measurement of cerebral **blood** flow and CMRO2 has been provided various useful knowledges on hemodynamics after ischemic cerebrovascular disease. Immediately after the occlusion, the ischemic lesion showed elevated oxygen extn. fraction (OEF), i.e., the misery perfusion which was defined as poor supply compared to demand for energy. However, one week after the onset, the OEF turned out to low value, this is, the luxury perfusion which was defined as excess supply compared to demand for energy. At the chronic stage one month after the onset, cerebral **blood** flow and CMRO2 were coupled. OEF is thus an indicator of the metabolic reserve. In addn., cerebral **blood** flow responsiveness to PaCO2 change will provide circulation reserve in cerebral vascular insufficiency. Degenerative disease can also be evaluated by cerebral **blood** flow and metab. using PET. Most of these diseases might be caused by neural transmission, or other degeneration, however, current PET technol. does not fully cover the individual specific target of these geneses. On the other hand, changes in cerebral **blood** flow and metab. will, although the non-primary cause of disease, be the most general, earliest symptoms. Combination with psychophysiol. or pharmaceutical perturbations will provide further possibility for early findings of these sub-clin. symptoms.

IT **50-99-7**, **Glucose**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(brain circulation and metab. measurement using positron emission tomog.)

L6 ANSWER 10 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1991:40020 CAPLUS
DN 114:40020
TI Polarized membrane expression of brush-border hydrolases in primary cultures of kidney proximal tubular cells depends on cell differentiation and is induced by dexamethasone
AU Ronco, P.; Antoine, M.; Baudouin, B.; Geniteau-Legendre, M.; Lelongt, B.; Chatelet, F.; Verroust, P.; Vandewalle, A.
CS Lab. Histol. Cytol. Pathol., Hop. Tenon, Paris, 75970, Fr.
SO J. Cell. Physiol. (1990), 145(2), 222-37
CODEN: JCLLAX; ISSN: 0021-9541
DT Journal
LA English
AB To analyze the influence of cell differentiation and the effects of hormones on the subcellular distribution of apical antigens of polarized epithelial cells, the localization of 3 brush border

hydrolases (neutral endopeptidase, aminopeptidase N, and dipeptidylpeptidase IV) in primary cultures of renal proximal tubule cells grown in various culture media was compared. The degree of cell differentiation modulated by medium compn. was estd. by measuring proximal functions, including **glucose** transport, specific enzymic activities, and parathormone responsiveness. In the dedifferentiated state obsd. in cells grown in 1% fetal calf serum (FCS)-supplemented medium, the 3 hydrolases are abnormally concd. in a cytoplasmic vesicle **compartment** with weak expression on both membrane domains. By contrast, in serum-free hormonally defined medium (DM: insulin, 5 .mu.g/mL; dexamethasone, 5 .times. 10-8M), which markedly enhances morphol. and functional cell differentiation, the distribution by hydrolases parallels that obsd. in the normal tubule. When added to the DM devoid of hormones, insulin has little polarizing effect, whereas dexamethasone dramatically increases the apical expression of the hydrolases, which then almost disappear from the basolateral membrane and cytoplasmic vesicular **compartments**. This glucocorticoid hormone augments the amt. of immunoreactive antigen detectable on the apical domain in paraformaldehyde-fixed cells but does not change the total enzymic activity. This suggests the presence in tubular cells of a dexamethasone-dependent polarizing machinery that requires de novo RNA and protein synthesis, and probably acts mainly by targeting a storage cytoplasmic pool of enzyme to the apical domain.

IT **9001-51-8, Hexokinase**
RL: BIOL (Biological study)
(of kidney proximal tubule cells, in culture, cell differentiation in relation to)

IT **50-99-7**, D-**Glucose**, biological studies
RL: BIOL (Biological study)
(transport of, by kidney proximal tubule cells in culture, cell differentiation in relation to)

L6 ANSWER 11 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1990:213395 CAPLUS
DN 112:213395
TI Michaelis-Menten constraints improved cerebral **glucose** metabolism and regional lumped constant measurements with [18F]fluorodeoxyglucose
AU Kuwabara, Hiroto; Evans, Alan C.; Gjedde, Albert
CS McConnell Brain Imaging Cent., Montreal Neurol. Inst., Montreal, PQ, H3A 2B4, Can.
SO J. Cereb. Blood Flow Metab. (1990), 10(2), 180-9
CODEN: JCBMDN; ISSN: 0271-678X
DT Journal
LA English
AB In the three-**compartment** model of transfer of native **glucose** and [18F]fluorodeoxyglucose (FDG) into brain, both

transport across the **blood**-brain barrier and phosphorylation by **hexokinase** can be described in the Michaelis-Menten equation. This permits the use of fixed transport (.tau. = K1*/K1) and phosphorylation (.phi. = k3*/k3) ratios and a common partition vol. (Vc = K1/k2) for tracer and **glucose**. By substituting transfer consts. of FDG for those of **glucose**, using .tau. and.phi., the lumped const. was detd. directly by positron tomog. The same constraints also eliminated k2* and k3* from the model, thus limiting the parameters to K* [equiv. to K1k3*/(k2* + k3*)], K1*, and the cerebral vascular vol. (Vo). In 6 healthy elderly men (aged 61 yr), time-activity records of cerebral cortical regions were analyzed with .tau. = 1.1 and .phi. = 0.3. The results were compared with those of the conventional FDG method. At 20 min, the goodness of fit by the new equation was as good as that of the conventional method at 45 min. The ests. obtained by the constrained method had stable coeffs. of variation. After 20 min, regional differences though steady decreases of K* and (k3*) were obsd. The decrease strongly suggested dephosphorylated of FDG-6-phosphate, particularly after 20 min. All ests. of variables with the constrained method were more accurate than those of the conventional method were more accurate than those of the conventional method, including the cerebral **glucose** metabolic rate itself, as well as physiol. more meaningful, particularly with respect to k2* and k3*.

IT **50-99-7**, **Glucose**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metab. of, by brain, fluorodeoxyglucose in detn. of, Michaelis-Menten constraints in)

L6 ANSWER 12 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1989:551975 CAPLUS
DN 111:151975
TI NADH and flavin fluorescence responses of starved yeast cultures to substrate additions
AU Siano, S. A.; Mutharasan, R.
CS Dep. Chem. Eng., Drexel Univ., Philadelphia, PA, 19104, USA
SO Biotechnol. Bioeng. (1989), 34(5), 660-70
CODEN: BIBIAU; ISSN: 0006-3592
DT Journal
LA English
AB Model expts. were performed with starved yeast (Saccharomyces cerevisiae) cultures in a batch reactor in order to develop a better understanding of NAD(P)H and flavin culture fluorescence. Fluorescence was monitored during aerobic-anaerobic-aerobic transitions and ethanol and **glucose** substrate addn. expts. Interpretations of the fluorescence responses obtained are provided, with consideration given to redox **compartmentation** and the formation of ethanol shortly after a **glucose** addn. An

anal. spectrofluorophotometer was interfaced to a personal computer and adapted to measure fluorescence in a bioreactor. This was achieved by the use of quartz fiber-optic waveguides to convert the right-angle cuvette geometry of the anal. spectrofluorophotometer to an open-ended fluorescence probe geometry, resulting in a flexible culture fluorescence app. Features of the app. include variable excitation and emission wavelengths, allowing for detection of NAD(P)H or flavin fluorescence, as well as small slit widths, a variable **sampling** rate, excitation and emission scanning capabilities, and good sensitivity.

IT **50-99-7**, **Glucose**, biological studies
RL: BIOL (Biological study)
(flavin and NADH fluorescence response to, in Saccharomyces cerevisiae, starvation in relation to)

IT **9001-18-7**
RL: PRP (Properties)
(fluorescence of, in Saccharomyces cerevisiae, substrate addn. effect on, starvation in relation to)

L6 ANSWER 13 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1989:151561 CAPLUS
DN 110:151561
TI **Blood glucose** partition and levels of glycolytic enzymes in erythrocytes and somatic tissues of penguins
AU Rosa, Rubens; Rodrigues, Edson; Bacila, Metry
CS Dep. Bioquim., Univ. Sao Paulo, Sao Paulo, Brazil
SO Comp. Biochem. Physiol., B: Comp. Biochem. (1989), 92B(2), 307-11
CODEN: CBPBB8; ISSN: 0305-0491
DT Journal
LA English
AB A comparative study was carried out on **blood glucose** partition and **glucose** metab. of Pygoscelidae penguins (Pygoscelis antarctica and P. papua) erythrocytes and somatic tissues. **Blood glucose** partition was established by assaying whole **blood** and **plasma glucose** in several individuals of the gentoo and chinstrap penguins. Almost all the whole **blood** sugar was **compartmentalized** at the **plasma** site, with the red **blood** cells being ineffective with regard to **glucose** metab. The levels of **hexokinase**, phosphoglucose isomerase, phosphofrutcokinase, fructose bisphosphate aldolase, glyceraldehyde phosphate dehydrogenase, phosphoglycerate kinase, phosphopyruvate hydratase (enolase), pyruvate kinase, .alpha.-glycerolphosphate dehydrogenase, and fructose bisphosphate phosphatase were estd. in the erythrocytes of both gentoo and chinstrap penguins, the same detns. being carried out also on the somatic tissues (leg muscle, breast muscle, heart muscle, liver, and brain) of the gentoo.

IT **50-99-7**, **Glucose**, biological studies

RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metab. of, by erythrocytes and organs of penguins)
IT **9001-51-8, Hexokinase**
RL: BIOL (Biological study)
(of erythrocytes and organs, of penguins)

L6 ANSWER 14 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1988:34501 CAPLUS
DN 108:34501
TI Assay for degradable substrates by electrochemical detection of redox species
IN McNeil, Calum Jack; Green, Monika Joanna; Hill, Hugh Allen Oliver
PA Genetics International, Inc., UK
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
PI WO 8604926 A1 19860828
DS W: AU, JP, US
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE
AI WO 86-GB95 19860221
PRAI GB 85-4522 19850221
DT Patent
LA English
AB In electrochem. detection of a redox species in a medium to det. the occurrence of a condition under which such species are produced or released, such prodn. or release is enabled by the digestion or disruption of a macromol. species. The invention provides a method of assay for a polymer, for a depolymerase, and for a labeled specific binding agent, in which a polymer is labeled with a component of a mediator/enzyme substrate system which remains inactive until released into soln. Electrodes in the medium det. the occurrence of a condition under which redox species are produced by the mediator/enzyme/substrate system or released into soln. Where the mediator activity of e.g. ferrocene to **glucose** oxidase is used, an amplification step is provided. Application to assay of polysaccharides, celluloses, pectins, galactosides, hyaluronic acids, lipids, peptides, nucleic acids, and immunochem. specific binding partners is indicated. 6-O-Ferrocene-amylose was prepd. (prepn. given) and 3.0 mg was suspended in 10 mL phosphate buffer for 2 min to make a suspension, which was placed in the **sample compartment** of a 2-**compartment**, 3-electrode electrochem. cell having a working vol. of 1 mL. After 10 min equilibration, a d.c. cyclic voltammogram taken at 0-650 mV vs. SCE showed no direct electrochem. at the working electrode. Amyloglucosidase 1 mL was added and incubation of the ferrocene-amylose complex with amyloglucosidase gave an increase in current with time due to the release of 6-O-ferrocenoylglucose from the complex. Adding **glucose** (50 .mu.L of a 1 M soln. in acetate buffer, pH 4.8) and **glucose** oxidase (50 .mu.L of a

3 mg/mL soln. in acetate buffer, pH 4.8) caused an increase in the anodic current from 110 to 285 nA.

IT **9028-53-9, Glucose dehydrogenase**
RL: ANST (Analytical study)
(in electrochem. assay of degradable substrates)

IT **50-99-7, Glucose**, biological studies
RL: BIOL (Biological study)
(in electrochem. assay of degradable substrates)

L6 ANSWER 15 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1984:507918 CAPLUS
DN 101:107918
TI Intracellular localization of rat kidney **hexokinase**. Evidence for an association with low density mitochondria
AU Parry, David M.; Pedersen, Peter L.
CS Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SO J. Biol. Chem. (1984), 259(14), 8917-23
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The subcellular location of **hexokinase** was investigated in rat kidney. Both sol. and particulate locations were indicated by differential centrifugation. The particulate form was predominant, representing .apprx.80% of the total activity. None of the activity was latent. D. gradient centrifugation followed by marker enzyme anal. revealed the presence of 2 populations of mitochondria with distinct densities. **Hexokinase** was assocd. primarily with the mitochondrial population having the lower d. Assocn. of **hexokinase** with brush border, **plasma** membrane, lysosomes, and endoplasmic reticulum was considered unlikely on the basis of d. gradient centrifugation and enzyme anal. About 95% of the **hexokinase** activity assocd. with the mitochondrial fraction could be released in sol. form by repeated incubations with **glucose** 6-phosphate. An incubation time of .apprx.4 min at 30.degree. was required to achieve a maximal solubilizing effect. Release was accomplished without disrupting the mitochondrial **compartments**. **Hexokinase** was released also by treatment of the mitochondrial fraction with increasing concns. of digitonin. This technique disrupted and differentially released the mitochondrial **compartments**. As obsd. with liver, but in contrast to that obsd. with tumor, the release of **hexokinase** from the mitochondrial fraction of kidney did not correlate with the release of enzymes known to mark the mitochondrial membranes or **compartments**. These studies provide the 1st crit. evidence about the subcellular location of **hexokinase** in kidney. They show that in this tissue **hexokinase** is assocd. primarily with low-d. mitochondria, a finding that adds credibility to the existence of this discrete population of mitochondria in

vivo. Significantly, this assocn. of **hexokinase** with kidney mitochondria appears unique in that its release on submitochondrial fractionation does not correlate with the release of known mitochondrial marker enzymes. These results are directly relevant to those cells in the kidney which utilize **glucose** as an energy source. It is suggested that the enhanced glycolytic capacity of these cells may be due, at least in part, to an assocn. of **hexokinase** with low-d. mitochondria.

IT **9001-51-8**
RL: PROC (Process)
(in kidney mitochondria, localization of)

L6 ANSWER 16 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1984:154534 CAPLUS
DN 100:154534
TI ATP-ADP-dependent phosphorylations of glycolysis metabolites, creatine and glycerol: their **compartition** and thermodynamic relationship in gastrocnemius muscle cell of exercised guinea pigs
AU Feraudi, M.; Kolb, J.; Hassel, Monika; Weicker, H.
CS Inst. Pathophysiol. Sports Med., Univ. Heidelberg, Heidelberg, Fed. Rep. Ger.
SO Arch. Int. Physiol. Biochim. (1983), 91(4), 351-60
CODEN: AIPBAY; ISSN: 0003-9799
DT Journal
LA English
AB The concns. of following metabolites were detd. in freeze-clamped gastrocnemius muscle **samples**: **glucose** 1-phosphate, **glucose** 6-phosphate, **glucose**, fructose 1,6-diphosphate, fructose 6-phosphate, D-glyceraldehyde 3-phosphate, dihydroxyacetone phosphate, phosphoenolpyruvate, pyruvate, glycerol 3-phosphate, glycerol, creatine phosphate, creatine, glycerate 3-phosphate, glycerate 2-phosphate, AMP, ADP, ATP, and inorg. phosphate. Within the limits of exptl. error, concn. homeostasis for this metabolites is founded at least in some cases on equil. between enzymic transformations. Discrepancies between const. mass ratios measured in this study and equil. consts. allow the free energy variation to keep creatine phosphate at high concn. to be calcd. For the phosphoglycerate mutase system, the equil. const. in controls and trained animals is unchanged and corresponds to that in vitro. Training hindered glycolysis and favored phosphorylation of creatine by glycerol 3-phosphate. Metabolites of the pyruvate kinase and **hexokinase** system cannot be homogeneously distribution in 1 space. The creatine kinase system is also sepd. from the **hexokinase** and pyruvate kinase system. A **compartition** of glycolytic process in gastrocnemius muscle seems to be inferred from these results.

IT **50-99-7**, biological studies **9001-51-8**

RL: BIOL (Biological study)
(of muscle, in exercise, **compartmentation** of)

L6 ANSWER 17 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1983:30334 CAPLUS
DN 98:30334
TI **Compartmentation** of **hexokinase** in human **blood** cells. Characterization of soluble and particulate enzymes
AU Rijksen, G.; Staal, G. E. J.; Beks, P. J.; Streefkerk, M.; Akkerman, J. W. N.
CS Dep. Haematol., State Univ. Hosp., Utrecht, 3500 CG, Neth.
SO Biochim. Biophys. Acta (1982), 719(3), 431-7
CODEN: BBACAQ; ISSN: 0006-3002
DT Journal
LA English
AB The isoenzyme distribution, kinetic properties, and intracellular localization of **hexokinase** (EC 2.7.1.1) were studied in erythrocytes, **blood** platelets, lymphocytes, and granulocytes. Sol. and particulate fractions were sepd. by a rapid d. centrifugation method after controlled digitonin-induced cell lysis. In lymphocytes and platelets, the major part of total activity was particle bound (78% and 88%, resp.). In granulocytes and erythrocytes, most of the **hexokinase** activity was found in the cytosol. All cell types, except granulocytes, contain mainly the type I isoenzyme. Platelets contain only type I **hexokinase**, whereas in lymphocytes, a minor amt. of type III is present in the sol. fraction (<10% of total activity). The major constituent of granulocytes is type III **hexokinase** (70-80% of total activity); the remaining 20-30% is type I **hexokinase**. Erythrocytes contain a multibanded type I **hexokinase**. The substrate affinities of the type I **hexokinase** do not differ significantly between the different cell types or between sol., bound, and solubilized fractions. Only sol. **hexokinase** from lymphocytes shows a slightly decreased apparent Km for **glucose**.
IT **9001-51-8**
RL: BIOL (Biological study)
(isoenzymes, of **blood** cells of human, localization and properties of)
IT **50-99-7**, reactions
RL: RCT (Reactant)
(reaction of, with **hexokinase** of human **blood** cells, kinetics of)

L6 ANSWER 18 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1980:634562 CAPLUS
DN 93:234562
TI Use of automatic methods for photometry and further development and

devices for performing these methods
IN Berthold, Fritz; Kolehmainen, Seppo; Tarkkanen, Veikko
PA Laboratorium Prof. Dr. Berthold, Fed. Rep. Ger.
SO Ger. Offen., 39 pp.
CODEN: GWXXBX
PI DE 2901919 19800724
AI DE 79-2901919 19790118
DT Patent
LA German
AB A method and automatic app. for photometric analyses are described which are intended to prevent disturbances in light measurement caused by exposing **samples** to light during reagent addns. In the method, successive addns. of reagents are made to a series of **samples** in a darkened **chamber**, the progress of the reaction is followed after each reagent addn. and the data are stored, and finally, from the values obtained in all the measuring periods, .gtoreq.1 unknown compd. in the **samples** may be calcd. by means of a computer program. Bioluminescence, chemiluminescence, fluorescence, or absorption measurements can be made, and various biol. compds. (nucleotides, enzymes, coenzymes, metabolites, etc.) can be quantitated.
IT **50-99-7**, analysis **9028-53-9**
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, by automatic analyzer with decreased light exposure on **sample**)

L6 ANSWER 19 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1979:570834 CAPLUS
DN 91:170834
TI Method for determination of enzyme activity
IN Takizawa, Koichi; Kaede, Kunio
PA Tateishi Electronics Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
PI JP 54097491 19790801 Showa
AI JP 78-4638 19780119
DT Patent
LA Japanese
AB Enzyme activities are electrochem. detd. in a reaction **chamber** which consisted of a ref. electrode and a reaction electrode coated with a porous polymer membrane contg. immobilized coenzymes. **Samples** and substrates are placed into the reaction **chamber** and the activity of enzyme is then detd. by measuring the elec. current produced from oxidn. or redn. of coenzymes. Thus, **glucose** and **samples** contg. **glucose dehydrogenase** were introduced into a reaction **chamber** which contained a ref. electrode and a reaction electrode coated with a cellulose or cellulose ester membrane on which NAD or NADH is immobilized, and an elec. potential

of 0.5-1.0 V was maintained. The enzyme activity was detd. by measuring the elec. current produced from oxidn. of NADH to NAD or redn. of NAD to NADH.

IT **9028-53-9**
RL: ANT (Analyte); ANST (Analytical study)
(detn. of, electrochem., coenzyme-coated reaction electrode for)

IT **50-99-7**, reactions
RL: RCT (Reactant)
(reaction of, with **glucose dehydrogenase**, electrochem. detn. of)

L6 ANSWER 20 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1979:3127 CAPLUS
DN 90:3127
TI Electrophoresis and demonstration of enzymic activities in Hevea brasiliensis latex
AU Jacob, J. L.; Nouvel, A.; Prevot, J. C.
CS Lab. Biochim. Physiol. Veg., Inst. Rech. Caoutchouc, Montpellier, Fr.
SO Rev. Gen. Caoutch. Plast. (1978), 55(582), 87-90
CODEN: RCPLA5; ISSN: 0035-3175
DT Journal
LA French
AB Enzymic activity in aq. exts. from the cytoplasm or organelles (leucoplasts and lysosomes) of H. brasiliensis latex was studied by polyacrylamide gel electrophoresis, using specific staining techniques to demonstrate the presence of reaction products. Nine dehydrogenases, 4 transferases, 4 hydrolases, 2 oxidases, 1 isomerase, and 1 carboxylase were detected. Four enzymes not previously reported in latex were demonstrated: galactose 6-phosphate dehydrogenase, leucine aminopeptidase, monoamine oxidase, and malic enzyme. The presence of fructokinase and a distinct **hexokinase**, previously reported from indirect evidence, was confirmed. Enzymic activity was **compartmentalized**. Only 5 enzymes were found both in cytoplasm exts. and in the organelles: malate dehydrogenase, **glucose** 6-phosphate isomerase, acid phosphatase, esterase, and peroxidase, and these enzymes differed structurally between **compartments**. Leucine aminopeptidase was found only in the organelles, and 4 of 6 enzymes localized in the organelles were hydrolases. Fifteen of the enzymes were found only in the cytoplasm, where the major part of rubber biosynthesis occurs. Multiple gel bonds appeared for 12 of the 21 enzymic activities demonstrated. To evaluate genetic implications, gel patterns for 4 such activities were compared for latex cytoplasm **fluid** from 5 different clonal origins. Differences occurred in no. and intensity of bands with esterase, peroxidase, and alc. dehydrogenase. In these cases the clone IAN 717, which came from Brazil, had more bands of greater intensity than the other clones,

which came from Asia. Phosphoglucomutase, however, gave 2 identical bands in all 5 clones.

IT **9001-51-8**
RL: BIOL (Biological study)
(isoenzymes, of latex of Hevea brasiliensis)

L6 ANSWER 21 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1968:484855 CAPLUS
DN 69:84855
TI **Hexokinase**, glucokinase, and **glucose**-6-phosphate dehydrogenase activities changes in various tissues of the rat during overheating
AU Kozlov, N. B.; Protchenko, O. A.
CS Smolensk. Med. Inst., Smolensk, USSR
SO Vop. Med. Khim. (1968), 14(4), 385-9
CODEN: VMDKAM
DT Journal
LA Russian
AB Rats were heated at 40-45.degree. until heat shock developed. Some rats were decapitated immediately after removal from the heat **chamber** and others were sacrificed later, and the brain, liver, heart, and skeletal muscles were studied. Heat shock was accompanied by an abrupt decrease of activity of glucokinase in the liver and a noticeable decrease of **hexokinase** activity in the skeletal muscles. **Hexokinase** activity in brain tissue and cardiac muscle was not noticeably changed. **Glucose** -6-phosphate dehydrogenase was unchanged in the liver and brain tissue during overheating but oxidn. of **glucose** 6-phosphate by pentose phosphate pathways was greatly decreased in the liver. Addn. to the incubation medium of **blood** serum of healthy animals led to inhibition of glucokinase activity of skeletal muscles and increase of glucokinase activity of the liver. **Glucose**-6-phosphate dehydrogenase activity of the liver was unchanged. **Blood** serum of overheated animals had less influence on glucokinase activity of skeletal muscles and liver than the **blood** serum of healthy animals. Glucokinase of the liver of overheated rats was less sensitive to the addn. of **blood** serum of healthy and overheated animals. 12 references.

L6 ANSWER 22 OF 22 CAPLUS COPYRIGHT 1998 ACS
AN 1968:1576 CAPLUS
DN 68:1576
TI Effects of decreased oxygen tension on cerebral circulation, metabolism, and function
AU Cohen, Peter J.
CS Walter Reed Army Med. Center, Washington, D. C., USA
SO Proc. Int. Symp. Cardiovasc. Respir. Eff. Hypoxia (1966), Volume 1965, 81-104

CODEN: 17CGAU
DT Conference
LA English
AB A review is presented of current knowledge of the fate of the brain during hypoxia. The profound effects of hypoxia upon cerebral circulation, metabolism, and function as well as the factors which may interact with diminished cerebral oxygenation are discussed. Functional changes occurring with hypoxia are described and appear to depend upon the relation of energy production to energy utilization. New data are presented on regional cerebral **blood** flow and the metabolic effects of hypoxia. The effect of hypoxia on regional cerebral **blood** flow was measured in 2 major **compartments** of the brain which are considered fast and slow, resp., in a hemodynamic sense. The rate of both total and regional cerebral perfusion was increased, and there was a change in the contributions of the numerous regional cerebral **blood** flows so that areas normally belonging to the slow **compartment** were perfused at rates closely related to that of the fast **compartment**. Accompanying changes in pCO2 modify this to an extent. Cerebral hypoxia is known to be accompanied by an increase in the level of lactate and inorg. phosphate and a decrease in the amts. of ATP and phosphocreatine. Studies of awake man inhaling 6.5-7.5% O2, with pCO2 held const., showed that for a drop to 34 mm. in the arterial O2 tension and to 27 mm. in the jugular venous O2 tension, cerebral **blood** flow increased from 45 to 77 ml./100 g./min., cerebral O2 consumption was unchanged, the rate of **glucose** utilization increased by 22%, lactate production increased 4-fold, the proportion of **glucose** metabolized aerobically decreased, and the proportion of **glucose** metabolized anaerobically increased. The augmented flux of **glucose** is probably regulated at the **hexokinase** and phosphofructokinase steps in the Embden-Meyerhof pathway and by the phosphorolysis of glycogen. 83 references.

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=> d his l7- ful; d 1-20 bib abs

     FILE 'USPATFULL' ENTERED AT 14:54:41 ON 26 OCT 1998
L7          310 SEA ABB=ON  PLU=ON  L5 AND (CHAMBER? OR COMPART?)
L8            1 SEA ABB=ON  PLU=ON  L7 AND OPTICAL READING
L9           91 SEA ABB=ON  PLU=ON  L7 AND OPTICAL
L10          91 SEA ABB=ON  PLU=ON  L9 AND (MEAS? OR DETERM? OR DETECT?
                OR DET## OR QUANT? OR CALCUL? OR CALC##)
L11          19 SEA ABB=ON  PLU=ON  L10 AND CYLIND?
L12          20 SEA ABB=ON  PLU=ON  L8 OR L11
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L12 ANSWER 1 OF 20 USPATFULL
AN 1998:39678 USPATFULL
TI Monoclonal antibody against an interferon-induced human protein in pure form
IN Horisberger, Michel Andre, Langgartenweg 12, 4123 Allschwil, Switzerland
Hochkeppel, Heinz-Kurt, Traugott Meyerstrasse 1, 4147 Aesch, Switzerland
Content, Jean, 5, Ave. Simonne, 1640 Rhode-St-Genese, Belgium
PI US 5739290 980414
AI US 95-444344 950518 (8)
RLI Division of Ser. No. US 94-258902, filed on 13 Jun 1994, now patented, Pat. No. US 5466585 which is a continuation of Ser. No. US 92-983177, filed on 30 Nov 1992, now abandoned which is a division of Ser. No. US 91-810580, filed on 19 Dec 1991, now patented, Pat. No. US 5198350 which is a continuation of Ser. No. US 90-497748, filed on 19 Mar 1990, now abandoned which is a continuation of Ser. No. US 87-37754, filed on 13 Apr 1987, now abandoned
PRAI GB 86-9162 860415
GB 86-25381 861023
DT Utility
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Basham, Daryl A.
LREP Ferraro, Gregory D.
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2014
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to purified proteins induced in human cells by interferon .alpha. or .beta., RNAs, DNAs and hybrid vectors coding for said proteins, hosts tranformed with such a hybrid vector, processes for the preparation and purification of these proteins, DNAs, vectors and hosts, monoclonal antibodies specific to these proteins, monoclonal antibody derivatives, hybridoma celll lines secreting these monoclonal antibodies specific to these proteins, and their derivatives in the qualitative and **quantitative determination** of these proteins, test kits containing the monoclonal antibodies, and pharmaceutical preparations containing said proteins. A protein of the invention shows antiviral properties ascribed to interferons and may be a valuable indicator of the cell response to an interferon therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 2 OF 20 USPATFULL
AN 1998:22107 USPATFULL
TI Method and device for specific binding assay

IN Yamauchi, Tadakazu, Saitama, Japan
Terasawa, Hideyuki, Saitama, Japan
PA Mochida Pharmaceutical Co., Ltd., Tokyo, Japan (non-U.S. corporation)
PI US 5723345 980303
AI US 95-495028 950627 (8)
PRAI JP 94-146865 940628
DT Utility
EXNAM Primary Examiner: Chin, Christopher L.
LREP Birch, Stewart, Kolasch & Birch, LLP
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 32 Drawing Figure(s); 29 Drawing Page(s)
LN.CNT 3621
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a general purpose specific binding assay method which has the advantages of highly accurate and quick measurements which exclude the effects of various factors that decrease reliability of the measured values, such as non-specific reactants in test **samples**, assay conditions and inactivation and the like changes in the activity of reagents. The present invention is further drawn to a specific binding assay device suitable for the practice thereof. The binding assay of the present invention is achieved by allowing a signal substance generator which takes part in a specific binding reaction and generates a signal substance, together with a liquid **sample**, to flow through a predetermined channel in a predetermined direction, thereby effecting generation of the specific binding reaction of a substance to be assayed to form a distribution of the signal substance generator in the channel in response to the concentration of the substance to be assayed, allowing the signal substance generator distributed in the channel to generate the signal substance, detecting the generated signal substance by a plurality of detection means arranged at different positions in the liquid flow direction, and arithmetically processing the plural detection results to minimize influence of other factors than the concentration of the substance to be assayed upon the assay result.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 3 OF 20 USPATFULL
AN 97:104347 USPATFULL
TI Methods and reagents for the rapid **determination** of glycated hemoglobin
IN Fiechtner, Michael D., Highland Park, IL, United States
Ramp, John M., Gurnee, IL, United States
England, Barbara J., Milwaukee, WI, United States
Annino, Mary J., Arlington Heights, IL, United States
Searcher : Shears 308-4994

PA Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)
PI US 5686316 971111
AI US 95-431398 950428 (8)
RLI Continuation of Ser. No. US 91-717558, filed on 19 Jun 1991, now abandoned
DT Utility
EXNAM Primary Examiner: Chin, Christopher L.
LREP Weinstein, David L.
CLMN Number of Claims: 20
ECL Exemplary Claim: 5
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1370
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is a rapid, continuous test for glycated hemoglobin using a non-equilibrium affinity binding method. Agarose beads derivatized with 3-aminophenylboronic acid specifically bind glycated hemoglobin. This solid phase is incorporated into a **sample** processor card, modified to mix and to separate the test solution from the solid phase prior to absorbance readings. Two absorbance readings are made on the test solution, one immediately after mixing the reagent/diluent with the **specimen**, and one after a significant amount of binding has occurred. A linear correlation between total glycated hemoglobin and hemoglobin A.sub.1c permits standardization and reporting of units equivalent to % hemoglobin A.sub.1c. Stable glycated hemoglobin solutions for use as standards in the assay, and a method for preparing the standards are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 4 OF 20 USPATFULL
AN 97:81114 USPATFULL
TI Devices and methods for the **measurement** of cellular biochemical processes
IN Cook, Neil David, Peterston-Super-Ely, United Kingdom
PA Amersham International plc, Buckinghamshire, England (non-U.S. corporation)
PI US 5665562 970909
WO 9426413 941124
AI US 95-373316 950117 (8)
WO 94-GB1040 940516
950117 PCT 371 date
950117 PCT 102(e) date
PRAI EP 93-303806 930517
DT Utility
EXNAM Primary Examiner: Gitomer, Ralph J.
LREP Wenderoth, Lind & Ponack
CLMN Number of Claims: 18

ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1634
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Apparatus and method for studying cellular processes comprise a vessel having a base including a layer comprising a scintillant substance and which is adapted for attachment and/or growth of cells. Cellular processes are examined by scintillation proximity assay using a reagent labelled with a radioisotope.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 5 OF 20 USPATFULL
AN 96:120804 USPATFULL
TI Method for preparing a glycated hemoglobin solution
IN Fiechtner, Michael D., Highland Park, IL, United States
Ramp, John M., Gurnee, IL, United States
England, Barbara J., Milwaukee, WI, United States
Annino, Mary J., Arlington Heights, IL, United States
PA Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)
PI US 5589393 961231
AI US 95-427508 950424 (8)
RLI Division of Ser. No. US 91-717558, filed on 19 Jun 1991, now abandoned
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Chin, Christopher L.
LREP Weinstein, David L.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1278
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is a rapid, continuous test for glycated hemoglobin using a non-equilibrium affinity binding method. Agarose beads derivatized with 3-aminophenylboronic acid specifically bind glycated hemoglobin. This solid phase is incorporated into a **sample** processor card, modified to mix and to separate the test solution from the solid phase prior to absorbance readings. Two absorbance readings are made on the test solution, one immediately after mixing the reagent/diluent with the **specimen**, and one after a significant amount of binding has occurred. A linear correlation between total glycated hemoglobin and hemoglobin A.sub.1c permits standardization and reporting of units equivalent to % hemoglobin A.sub.1c. Stable glycated hemoglobin solutions for use as standards in the assay, and a method for preparing the standards are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 6 OF 20 USPATFULL
AN 95:107897 USPATFULL
TI Cuvette
IN Nilsson, Sven-Erik, Dobeliusvagen 39, SE-256 54 Helsingborg, Sweden
Lilja, Jan, Sodra Brunnsvagen 63, SE-256 54 Helsingborg, Sweden
PI US 5472671 951205
AI US 94-196640 940214 (8)
DCD 20110215
RLI Continuation-in-part of Ser. No. US 91-768321, filed on 17 Oct 1991, now patented, Pat. No. US 5286454
PRAI SE 89-1518 890426
DT Utility
EXNAM Primary Examiner: Warden, Robert J.; Assistant Examiner: Tran, Hien
LREP Kane, Dalsimer, Sullivan et al.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 451
AB The present invention is a cuvette provided with at least two cavities. The first or inlet cavity takes up **fluid** by capillary action. The second cavity is a reception cavity which communicates with the first cavity through a first channel. The second cavity preferably is constructed and arranged to not exert a capillary action, although it could be constructed to exert a capillary action. The first channel which is in **fluid** communication with the first cavity and the second cavity exerts a non-capillary and non-spontaneous **fluid** transporting function that is operative only under an external influence, such as by application of a centrifugal force on the cuvette. The cuvette is well suited for a variety of analyses, particularly upon whole **blood**.

L12 ANSWER 7 OF 20 USPATFULL
AN 95:101119 USPATFULL
TI Interferon-induced human protein in pure form, monoclonal antibodies thereto, and test kits containing these antibodies
IN Horisberger, Michel A., Allschwil, Switzerland
Hochkeppel, Heinz-Kurt, Aesch, Switzerland
Content, Jean, Rhode-St-Genese, Belgium
PA Ciba-Geigy Corporation, Ardsley, NY, United States (U.S. corporation)
PI US 5466585 951114
AI US 94-258902 940613 (8)
RLI Continuation of Ser. No. US 92-983177, filed on 30 Nov 1992, now

abandoned which is a division of Ser. No. US 91-810580, filed on 19 Dec 1991, now patented, Pat. No. US 5198350 which is a continuation of Ser. No. US 90-497748, filed on 19 Mar 1990, now abandoned which is a continuation of Ser. No. US 87-37754, filed on 13 Apr 1987, now abandoned

PRAI GB 86-9162 860415
GB 86-25381 861023

DT Utility

EXNAM Primary Examiner: Martinell, James

LREP Elmer, James Scott

CLMN Number of Claims: 19

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 1986

AB The invention relates to purified proteins induced in human cells by interferon .alpha. or .beta., RNAs, DNAs and hybrid vectors coding for said proteins, hosts transformed with such a hybrid vector, processes for the preparation and purification of these proteins, DNAs, vectors and hosts, monoclonal antibodies specific to these proteins, monoclonal antibody derivatives, hybridoma cell lines secreting these monoclonal antibodies, the use of the monoclonal antibodies and their derivatives in the qualitative and **quantitative determination** of these proteins, test kits containing the monoclonal antibodies, and pharmaceutical preparations containing said proteins. A protein of the invention shows antiviral properties ascribed to interferons and may be a valuable indicator of the cell response to an interferon therapy.

L12 ANSWER 8 OF 20 USPATFULL

AN 94:3537 USPATFULL

TI Liposomal delivery system with photoactivatable triggered release

IN Thompson, David H., 12602 NW. Barnes Rd. Apt. 6, Portland, OR, United States 97229
Anderson, Valerie C., 1862 Woodland Ter., Lake Oswego, OR, United States 97034

PI US 5277913 940111

AI US 91-756504 910909 (7)

DT Utility

EXNAM Primary Examiner: Page, Thurman K.; Assistant Examiner: Kishore, G. S.

LREP Klarquist, Sparkman, Campbell, Leigh & Whinston

CLMN Number of Claims: 27

ECL Exemplary Claim: 1

DRWN 17 Drawing Figure(s); 9 Drawing Page(s)

LN.CNT 1162

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A triggered release liposomal delivery system is disclosed that selectively releases its contents in response to illumination or

reduction in pH. The liposomes contain an amphipathic lipid, such as a phospholipid, having two chains derived from fatty acid that allow the lipid to pack into a bilayer structure. One or both of the alkyl chains contains a vinyl ether functionality that is cleaved by reactive oxygen species (ROS) or acid. A photosensitizer is incorporated into the liposomal cavity or membrane, and produces ROS or acid when illuminated to cleave the vinyl ether functionality and disrupt the liposomal membrane to release the vesicle contents. The lipid is preferably a plasmalogen, for example ##STR1## wherein R.sub.1 and R.sub.2 are each long chain hydrocarbons containing 12-24 carbons.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 9 OF 20 USPATFULL
AN 93:24826 USPATFULL
TI Interferon-induced human protein in pure form, monoclonal antibodies thereto and test kits containing these antibodies
IN Horisberger, Michel A., Allschwil, Switzerland
Hochkeppel, Heinz-Kurt, Aesch, Switzerland
Content, Jean, Rhode-St-Genese, Belgium
PA Ciba-Geigy Corporation, Ardsley, NY, United States (U.S. corporation)
PI US 5198350 930330
AI US 91-810580 911219 (7)
RLI Continuation of Ser. No. US 90-497748, filed on 19 Mar 1990, now abandoned which is a continuation of Ser. No. US 87-37754, filed on 13 Apr 1987, now abandoned
PRAI GB 86-9162 860415
GB 86-25381 861023
DT Utility
EXNAM Primary Examiner: Martinell, James
LREP Villamizar, JoAnn
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1842
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to purified proteins induced in human cells by interferon .alpha. or .beta., RNAs, DNAs and hybrid vectors coding for said proteins, hosts transformed with such a hybrid vector, processes for the preparation and purification of these proteins, DNAs, vectors and hosts, monoclonal antibodies specific to these proteins, monoclonal antibody derivatives, hybridoma cell lines secreting these monoclonal antibodies, the use of the monoclonal antibodies and their derivatives in the qualitative and **quantitative determination** of these proteins, test kits containing the monoclonal antibodies, and pharmaceutical preparations containing said proteins. A protein of the invention

shows antiviral properties ascribed to interferons and may be a valuable indicator of the cell response to an interferon therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 10 OF 20 USPATFULL
AN 92:31780 USPATFULL
TI Analytical test device and the use thereof
IN Adler, Stanford L., Warwick, NY, United States
Campisi, John, Riverside, CT, United States
Leong, Koon-Wah, Ossining, NY, United States
PA Technicon Instruments Corporation, Tarrytown, NY, United States (U.S. corporation)
PI US 5106758 920421
AI US 88-282657 881212 (7)
DT Utility
EXNAM Primary Examiner: Warden, Robert J.; Assistant Examiner: Collins, Laura E.
LREP Greenman, Jeffrey M.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 15 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 898
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The analytical device includes a base adapted to support a porous member having dispersed throughout at least one **analyte** responsive substance which undergoes a **detectable** response to the presence and concentration of the **analyte** in the test **sample**. A cover integral with the base is provided with a reaction well adapted for storing a dried reaction component and for receiving the liquid test **sample** deposited on the analytical device whereby the liquid test **sample** and the reaction component form a liquid reaction mixture therein. A passage is included in the reaction well for selectively transferring the reaction mixture therethrough. This passage is normally spaced from the porous member and effectively closed to transfer therethrough of the reaction mixture, and is selectively movable toward the porous member to effectively open to passage therethrough of the reaction mixture whereby the **analyte** responsive substance will undergo a **detectable** response to the **analyte** present in the liquid reaction mixture. The base is provided with a suitable viewing port to permit monitoring of the **detectable** response generated as a result of the at least one **analyte** responsive substance interacting with the **analyte** in the test **sample**. Such a **detectable** response may be **detected** by appropriate photometric or colorimetric apparatus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 11 OF 20 USPATFULL
AN 91:86794 USPATFULL
TI Affinity matrices of modified polysaccharide supports
IN Hou, Kenneth C., Glastonbury, CT, United States
Liao, Tung-Ping D., Missouri City, TX, United States
Rohan, Robert, Columbia, CT, United States
PA Cuno Inc., Meridan, CT, United States (U.S. corporation)
PI US 5059654 911022
AI US 89-311498 890216 (7)
RLI Continuation-in-part of Ser. No. US 88-154815, filed on 11 Feb 1988, now abandoned which is a continuation-in-part of Ser. No. US 87-130186, filed on 8 Dec 1987, now abandoned which is a continuation-in-part of Ser. No. US 87-13512, filed on 27 Jan 1987, now abandoned which is a continuation-in-part of Ser. No. US 84-656922, filed on 2 Oct 1984, now patented, Pat. No. US 4639513 which is a continuation-in-part of Ser. No. US 84-576448, filed on 2 Feb 1984, now patented, Pat. No. US 4663163 which is a continuation-in-part of Ser. No. US 83-466114, filed on 14 Feb 1983, now abandoned
DT Utility
EXNAM Primary Examiner: Nutter, Nathan M.
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 3382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to a modified polysaccharide material which comprises: (1) polysaccharide covalently bonded to a synthetic polymer; (2) the synthetic polymer being made from (a) a polymerizable compound which is capable of being covalently coupled directly or indirectly to said polysaccharide, and (b) one or more polymerizable compounds containing (i) a chemical group capable of causing the covalent coupling of the compound (b) to an affinity ligand or a biologically active molecule or (ii) a hydrophobic compound.

The invention is also directed to devices for the chromatographic separation of at least two components of a mixture comprising the modified polysaccharide material of the invention, wherein the device is configured for radial or tangential flow.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 12 OF 20 USPATFULL
AN 91:62700 USPATFULL
TI Analysis by sensor placement in recprocating flow
IN Liffmann, Stanley M., Andover, MA, United States

Qureshi, Humayun, Wayland, MA, United States
Czaban, John D., Beverly, MA, United States
PA Apec, Inc., Danvers, MA, United States (U.S. corporation)
PI US 5037737 910806
AI US 88-213258 880629 (7)
DT Utility
EXNAM Primary Examiner: Lacey, David L.; Assistant Examiner: Waack, J. D.
LREP Wolf, Greenfield & Sacks
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 724
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method and apparatus for analyzing a **sample** to **determine** an **analyte** content has a sensor for sensing the **analyte** positioned between a first and second transducer. The **analyte** or derivatives thereof are reciprocated between the first and second transducer to reciprocate at the sensor and allow instantaneous sensing of an indication of the **analyte**'s presence at a point between the first and second transducer. Rapid analysis can be carried out in many different systems including for example where the transducers are enzyme reactors and the **analytes** are body metabolites.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 13 OF 20 USPATFULL
AN 90:80143 USPATFULL
TI Photoresponsive electrode for **determination** of redox potential
IN Hafeman, Dean, Hillsborough, CA, United States
PA Molecular Devices Corporation, Palo Alto, CA, United States (U.S. corporation)
PI US 4963815 901016
AI US 87-72168 870210 (7)
DT Utility
EXNAM Primary Examiner: Eisenzopf, Reinhard J.; Assistant Examiner: Mueller, Robert W.
LREP Allegretti & Witcoff
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 2328
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Devices and methods are provided for **determining** the presence and amount of an **analyte** by **measuring** a redox potential-modulated photoinducing signal from a

photoresponsive element. Further devices and methods are provided for **determining** the presence and amount of an **analyte** by **measuring** a redox potential, pH or ion modulated photoinduced signal from a photoresponsive element, where one signal is a constant system and the other signal(s) is a variable system. The constant system signal is used to standardize the variable system signal. Various protocols may be employed where an **analyte** may be directly or indirectly coupled to a redox couple, a pH or ion system for **detection**. The latter devices employ a photoresponsive element having a medium contacting surface, which is partially covered with an electronically conducting layer and partially covered with a protective insulative layer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 14 OF 20 USPATFULL
AN 85:19413 USPATFULL
TI Method for culturing cells or tissues
IN Rose, Sam, Eggertsville, NY, United States
PA Bio-Response, Inc., Wilton, CT, United States (U.S. corporation)
PI US 4508819 850402
AI US 82-380960 820521 (6)
RLI Continuation of Ser. No. US 81-287032, filed on 27 Jul 1981 which is a continuation of Ser. No. US 79-59497, filed on 23 Jul 1979, now abandoned which is a division of Ser. No. US 77-851744, filed on 15 Nov 1977, now patented, Pat. No. US 4189470 which is a division of Ser. No. US 75-549985, filed on 11 Feb 1975, now patented, Pat. No. US 4064006 which is a division of Ser. No. US 73-349330, filed on 9 Apr 1973, now patented, Pat. No. US 3964467 which is a continuation-in-part of Ser. No. US 73-328048, filed on 30 Jan 1973, now patented, Pat. No. US 3857393 which is a division of Ser. No. US 71-136467, filed on 22 Apr 1971, now patented, Pat. No. US 3719182
DT Utility
EXNAM Primary Examiner: Rosen, Sam
LREP Kenyon & Kenyon
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2043
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed herein is a process for the in vitro culturing of cells or tissues such that the cells or tissues grow, behave and function in substantially the manner such cells or tissues grow, behave and function within the body, wherein the cells or tissues are contacted or bathed with fresh flowing cell-free lymph. The cell-free lymph may be obtained directly from a living host by removing lymph from the host, rendering the lymph cell-free by

suitable means and then using the cell-free lymph as the culture medium for cells or tissues of either the same or different species as that of the host. The cells or tissues preferably are contacted with the cell-free lymph in a **chamber** in which the cells or tissues are exposed either directly to the flowing lymph or to components thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 15 OF 20 USPATFULL
AN 82:49566 USPATFULL
TI Method and apparatus for the **determination** of substances in biological solutions by differential pH **measurement**
IN Luzzana, Massimo, Via Olgettina 60, Milan, Italy
PI US 4353867 821012
AI US 80-192204 800930 (6)
DT Utility
EXNAM Primary Examiner: Serwin, Ronald E.
LREP Shlesinger, Arkwright, Garvey & Dinsmore
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 649
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method and apparatus for the **determination** of the concentration of certain chemical substances such as **glucose**, urea, enzymes, etc. in biological solutions such as **blood**, serum, urine, using a technique of differential pH **measurement** using two pH electrodes, and **measuring** pH changes in the solutions after the addition of specific reagents, the concentration being **determined** according to the formula

[**sample**]=FCAL.times.(.DELTA.pH.sub.c -.DELTA.pH.sub.b -.DELTA.pH.sub.o)

wherein [**sample**] is the concentration of the substance in the **sample**, FCAL is a calibration factor, .DELTA.pH.sub.c is the **measured** pH of the reacted **sample**, .DELTA.pH.sub.b is a pH correction for the reaction initiating compound, and .DELTA.pH.sub.o is a correction for background pH changes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 16 OF 20 USPATFULL
AN 81:23316 USPATFULL
TI Clinical analytical system
IN Natelson, Samuel, 925 Southgate Rd., Knoxville, TN, United States

37914
PI US 4264560 810428
AI US 79-106550 791226 (6)
DT Utility
EXNAM Primary Examiner: Serwin, Ronald
LREP Oujevolk, George B.
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 960
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An arrangement for chemical analysis of a small **quantity** of **sample** wherein a **specimen** of a small size is passed through a porous distribution first medium onto a reagent-containing second medium is disclosed. The reagent-containing second medium is a thin, flat, liquid-impervious medium. A reagent is encapsulated upon the second medium as a flat, liquid-phase surface. The first and second mediums are so arranged and disposed that when firmly pressed together, the encapsulated liquid reagent will be liberated and the **specimen** will be distributed through the first medium onto the liquid-phase liberated reagent where the subsequent reaction of the liquid-phase reagent and the **specimen** can then be identified by reading means.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 17 OF 20 USPATFULL
AN 81:1060 USPATFULL

fluid free of lymph cells is disclosed. The lymph is taken from a subject (e.g., human or animal) by means of a thoracic duct fistula by raising the pressure of the central venous system in order that the pressure is above the atmospheric pressure of the thoracic duct. Lymph is separated into cells and lymph **fluid** containing antibodies. Cells are returned to the subject intravenously. Lymph **fluid** is mixed with substrate particles attached with specific antigens. An antibody-antigen-substrate particle complex is formed and removed. Subsequently, specific antibody can be split from the specific antigens attached to a substrate particle. The antigen-attached substrate particles can then be re-used. Specific antibody can be introduced into another subject or used for other purposes in biology, in chemistry, and in veterinary and clinical medicine.

CAS INDEXIN VAILABLE FOR THIS PATENT.

L12 ANSWER 1 TFULL
AN 77:6640
TI Method f s mass in vitro suspension culture of cells
IN Rose, Sam, e, NY, United States
PA Bio-Respons ilton, CT, United States (U.S. corporation)
PI US 4064006
AI US 75-549085 1 (5)
RLI Division of Ser o. US 73-349330, filed on 9 Apr 1973, now patented, Pat. N . US 3964467 which is a continuation-in-part of Ser. No. US 73-328048, filed on 30 Jan 1973, now patented, Pat. No. US 3857393 which is a division of Ser. No. US 71-136476, filed on 22 Apr 1971, now patented, Pat. No. US 3179182
DT Utility
EXNAM Primary Examiner: Rosen, Sam
LREP Kenyon & Kenyon, Reilly, Carr & Chapin
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2119
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process for augmenting production of a specific antibody from a patient including the steps of administering a specific antigen to the patient to cause production of the specific antibody, or choosing a subject containing an antigen, e.g., cancer, or one already immunized or making antibodies to an endogenous or exogenous antigen, performing a thoracic duct fistula on the patient, raising the central venous pressure of the patient for example, by pericordial tamponde, and collecting lymph from the fistula, centrifuging the collected lymph to separate the lymph cells therein from the lymph **fluid**, and to form a thin elongated layer of the separated cells, treating the thin

elongated layer of lymph cells to assure that they are substantially free of the specific antibody produced, dispersing the cells in a physiologically balanced saline solution and returning the dispersed cells and solution to the patient intravascularly, and giving appropriate replacement therapy to maintain normal control and health of all other systems in the body, the replacement therapy comprising **fluids** which can be of several kinds, but having in common that they are substantially free of the specific antibody.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L12 ANSWER 20 OF 20 USPATFULL
AN 76:34385 USPATFULL
TI Methods and apparatus for augmentation of the production of anti-bodies in animals and humans and the collection thereof
IN Rose, Sam, Eggertsville, NY, United States
PA BIO Response Inc., Scarborough, NY, United States (U.S. corporation)
PI US 3964467 760622
AI US 73-349330 730409 (5)
DCD 19900306
RLI Continuation-in-part of Ser. No. US 73-328048, filed on 30 Jan 1973, now Defensive Publication No. which is a division of Ser. No. US 71-136476, filed on 22 Apr 1971, now patented, Pat. No. US 3179182
DT Utility
EXNAM Primary Examiner: Truluck, Dalton C.
CLMN Number of Claims: 45
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2276
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process for augmenting production of a specific antibody from a patient including the steps of administering a specific antigen to the patient to cause production of the specific antibody, or choosing a subject containing an antigen, e.g., cancer, or one already immunized or making antibodies to an endogenous or exogenous antigen, performing a thoracic duct fistula on the patient, raising the central venous pressure of the patient for example, by pericordial tamponde, and collecting lymph from the fistula, centrifuging the collected lymph to separate the lymph cells therein from the lymph **fluid**, and to form a thin elongated layer of the separated cells, treating the thin elongated layer of lymph cells to assure that they are substantially free of the specific antibody produced, dispersing the cells in a physiologically balanced saline solution and returning the dispersed cells and solution to the patient intravascularly, and giving appropriate replacement therapy to

maintain normal control and health of all other systems in the body, the replacement therapy comprising **fluids** which can be of several kinds, but having in common that they are substantially free of the specific antibody.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his l13-; d 1-43 bib abs

(FILE 'BIOSIS, MEDLINE, EMBASE, LIFESCI, BIOTECHDS, WPIDS, CONFSCI, DISSABS, SCISEARCH, JICST-EPLUS, PROMT' ENTERED AT 15:42:44 ON 26 OCT 1998)
L13 81 S L6
L14 43 DUP REM L13 (38 DUPLICATES REMOVED)

L14 ANSWER 1 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 1998339531 EMBASE
TI Evaluation of the Sakura GL II, a novel **glucose** analyzer intended for point-of-care monitoring.
AU Kuhn-Velten W.N.; Susanto F.; Reinauer H.
CS Prof. W.N. Kuhn-Velten, Diabetes-forschungsinstitut, Heinrich-Heine-Universitat, Abteilung fur Klinische Biochemie, Auf'm Hennekamp 65, D-40225 Dusseldorf, Germany
SO LaboratoriumsMedizin, (1998) 22/9 (484-488).
Refs: 9
ISSN: 0342-3026 CODEN: LABOD3
CY Germany
DT Journal; Article
FS 003 Endocrinology
027 Biophysics, Bioengineering and Medical Instrumentation
029 Clinical Biochemistry
LA English
SL English; German
AB The performance and practicability of an improved **glucose** analyzer using the immobilized **glucose** oxidase/H2O2 electrode technology, the Sakura GL II, was evaluated and compared to a fixed-time kinetic **hexokinase** method running on an Eppendorf Epos 5060 analyzer. Since the Sakura GL II measured **glucose** in the **plasma compartment**, whereas the Epos 5060 used hemolysates, results for 294 capillary **blood samples** were on average 11.3% higher with the Sakura GL II than with the comparison method. **Glucose** concentrations in serum **samples** certified for quality control surveys, however, were on average 8% lower with the immobilized **glucose** oxidase than with the **hexokinase** method. Mean within-run and between-day

imprecisions (coefficient of variation) were 3.9 and 3.7% for the Sakura GL II and 1.7 and 3.0% for the Epos 5060 (n = 10). Since the Sakura GL II was assessed as very user-friendly, it appeared suitable for point-of-care **glucose** monitoring in the **plasma compartment** of capillary **blood samples**.

L14 ANSWER 2 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1
AN 98:183015 BIOSIS
DN 01183015
TI **Glucose** metabolism in human malignant gliomas measured quantitatively with PET, 1-(C-11)**glucose** and FDG: Analysis of the FDG lumped constant.
AU Spence A M; Muzi M; Graham M M; O'Sullivan F; Krohn K A; Link J M; Lewellen T K; Lewellen B; Freeman S D; Berger M S; Ojemann G A
CS Dep. Neurol., Box 356465, University Washington, Seattle, WA 98195, USA
SO Journal of Nuclear Medicine 39 (3). 1998. 440-448. ISSN: 0161-5505
LA English
AB Calculation of the **glucose** metabolic rate (MRGlc) in brain with PET and 2-(18F)fluoro-2-deoxy-D-**glucose** (FDG) requires knowing the rate of uptake of FDG relative to **glucose** from **plasma** into metabolite pools in the tissue. The proportionality factor for this is the FDG lumped constant (LC-FDG), the ratio of the volumes of distribution of FDG and **glucose** multiplied by the **hexokinase** phosphorylation ratio for the two hexoses, Km-Glc cntdot Vm-FDG/Km-FDG cntdot Vm-Glc. MRGlc equals the FDG metabolic rate (MRFDG) divided by the LC-FDG, i.e., MRGlc = MRFDG/LC-FDG and LC-FDG = MRFDG/MRGlc. This investigation tested the hypothesis that LC-FDG is significantly higher in gliomas than it is in brain uninvolved with tumor. Methods: We imaged 40 patients with malignant gliomas with 1-(11C)**glucose** followed by FDG. The metabolic rates MRGlc and MRFDG were estimated for glioma and contralateral brain regions of interest by an optimization program based on three-**compartment**, four-rate constant models for the two hexoses. Results: The LC-FDG, estimated as MRFDG/MRGlc, in gliomas was 1.40 +- 0.46 (mean +- s.d.; range = 0.72-3.10), whereas in non-tumor-bearing contralateral brain, it was 0.86 +- 0.14 (range = 0.61-1.21) (p lt 0.001, glioma versus contralateral brain). Conclusion: These data strongly suggest that the glioma LC-FDG exceeds that of contralateral brain, that quantitation of the glioma MRGlc with FDG requires knowing the LC-FDG specific for the glioma and that the LCFDG of normal brain is higher than previously reported estimates of about 0.50. 2-Fluoro-2-deoxy-D-**glucose**/PET studies in which glioma **glucose** metabolism is calculated by the autoradiographic approach with normal brain rate constants and LC-FDG will overestimate glioma MRGlc, to the extent that the glioma LC-FDG exceeds the normal brain LC-FDG. "Hot spots" visualized in FDG/PEr studies of gliomas represent regions where MRGlc, LC-FDG or

their product is higher in glioma than it is in uninvolved brain tissue.

L14 ANSWER 3 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.DUPLICATE 2
AN 1998314870 EMBASE
TI An overview of muscle **glucose** uptake during exercise: Sites of regulation.
AU Wasserman D.H.; Halseth A.E.
CS Dr. D.H. Wasserman, Light Hall Dept. of Molec. Physiol., Vanderbilt Univ. School of Medicine, Nashville, TN 37232, United States. david.wasserman@mcmail.vanderbilt.edu
SO Advances in Experimental Medicine and Biology, (1998) 441/- (1-16). Refs: 122
ISSN: 0065-2598 CODEN: AEMBAP
CY United States
DT Journal; Article
FS 002 Physiology
LA English
SL English
AB The uptake of **blood glucose** by skeletal muscle is a complex process. In order to be metabolized, **glucose** must travel the path from **blood** to interstitium to intracellular space and then be phosphorylated to **glucose** 6- phosphate (G6P). Movement of **glucose** from **blood** to interstitium is determined by skeletal muscle **blood** flow, capillary recruitment and the endothelial permeability to **glucose**. The influx of **glucose** from the interstitium to intracellular space is determined by the number of **glucose** transporters in the sarcolemma and the **glucose** gradient across the sarcolemma. The capacity to phosphorylate **glucose** is determined by the amount of skeletal muscle **hexokinase** II, **hexokinase** II **compartmentalization** within the cell, and the concentration of the **hexokinase** II inhibitor G6P. Any change in **glucose** uptake occurs due to an alteration in one or more of these steps. Based on the low calculated intracellular **glucose** levels and the higher affinity of **glucose** for phosphorylation relative to transport, **glucose** transport is generally considered rate-determining for basal muscle **glucose** uptake. Exercise increases both the movement of **glucose** from **blood** to sarcolemma and the permeability of the sarcolemma to **glucose**. Whether the ability to phosphorylate **glucose** is increased in the working muscle remains to be clearly shown. It is possible that the accelerated **glucose** delivery and transport rates during exercise bias regulation so that muscle **glucose** phosphorylation exerts more control on muscle **glucose** uptake. Conditions that alter **glucose** uptake during exercise, such as increased NEFA concentrations, decreased oxygen

availability and adrenergic stimulation, must work by altering one or more of the three steps involved in **glucose** uptake. This review describes the regulation of **glucose** uptake during exercise at each of these sites under a number of conditions, as well as describing muscle **glucose** uptake in the post-exercise state.

L14 ANSWER 4 OF 43 DISSABS COPYRIGHT 1998 UMI Company
AN 1998:2776 DISSABS Order Number: AAR9803275
TI TESTING THE ASSUMPTIONS OF THE USE OF ACETAMINOPHEN GLUCURONIC ACID AS A PROBE OF THE INTRAHEPATIC UDP-**GLUCOSE** POOL USED FOR GLYCOGEN SYNTHESIS (CARBOHYDRATE METABOLISM)
AU LETSCHER, AMY ELIZABETH [PH.D.]; HELLERSTEIN, MARC K. [adviser]
CS UNIVERSITY OF CALIFORNIA, BERKELEY (0028)
SO Dissertation Abstracts International, (1997) Vol. 58, No. 8B, p. 4152. Order No.: AAR9803275. 194 pages.
DT Dissertation
FS DAI
LA English
AB Use of urinary acetaminophen-glucuronic acid as a probe of the intrahepatic UDP-**glucose** pool used for liver glycogen synthesis assumes that the enrichment in the urinary glucuronide is representative of the enrichment of the intrahepatic UDP-**glucose** pool that is used for liver glycogen synthesis. This assumption was tested in either fasted or fasted and intravenous (IV) **glucose** (10-15 or 30 mg/kg/min), IV fructose (10-15 mg/kg/min) or IV glycerol (10-15 mg/kg/min) refed male Sprague Dawley rats given a nine hour constant IV infusion of acetaminophen, (1-$\sp3$H) -**glucose**, and (2-$\sp{13}$C) -glycerol. Using the mass isotopomer distribution analysis (MIDA) technique, the triose phosphate precursor pool enrichment, p, and the gluconeogenic fraction, f, were determined and compared in **blood glucose**, liver glycogen or urinary acetaminophen-glucuronic acid.

The p measured using urinary acetaminophen-glucuronic acid was very similar to the p measured by the liver glycogen during IV **glucose** (r =.839), IV fructose (r$\sp2$ =.910), and IV glycerol (r$\sp2$ =.859) refeeding, for an overall correlation of r$\sp2$ =.837 (p $<$ 0.0001, N = 30). When the f measured using the urinary glucuronide was compared to the f measured using liver glycogen following IV **glucose**, IV fructose, and IV glycerol refeeding, a strong overall correlation of r$\sp2$ =.827 (p $<$ 0.0001, N = 30), was obtained. The mean f in urinary acetaminophen-glucuronic acid was consistently approximately 10% higher, though not significantly different, than the f in glycogen, indicating the presence of pre-existing glycogen. Similarly, the p measured in **plasma glucose** was significantly correlated to the p measured in urinary acetaminophen-glucuronic acid, with an overall correlation of r$\sp2$ = 0.907 during

refeeding and fasting experiments. Thus no evidence supporting the **compartmentalization** of either the intrahepatic UDP-**glucose**, **glucose** 6-phosphate or triose phosphate pools was obtained. These results suggest that acetaminophen-glucuronic acid can be used as a non-invasive probe of the intrahepatic UDP-**glucose** pool used for glycogen synthesis in vivo.

During progressive fasting of between 2 and 28 hours, hepatic **glucose** production (HGP) fell by over 85%, primarily due to the significant decrease in hepatic glycogenolysis, as the gluconeogenic contribution to HGP remained constant. IV **glucose** refeeding at either 10-15 or 30 mg/kg/min decreased HGP by significantly decreasing the absolute gluconeogenic contribution to HGP by 53 and 33%, respectively. Refeeding fasted rats with a substantial gluconeogenic precursor load of either IV glycerol or IV fructose at 10-15 mg/kg/min significantly increased HGP over their fasting values by 38 and 93%, respectively. The increase in HGP was primarily due to the significant (p $<$ 0.0001) increase in absolute gluconeogenesis. The increase in the non-gluconeogenic contribution to HGP observed following IV fructose may indicate that as much as 24 $\pm$ 9% of the infused fructose load was directly converted to fructose 6-phosphate within the liver by the action of **hexokinase**.

L14 ANSWER 5 OF 43 MEDLINE
AN 97153407 MEDLINE
DN 97153407
TI High-fat feeding impairs insulin-stimulated GLUT4 recruitment via an early insulin-signaling defect.
AU Zierath J R; Houseknecht K L; Gnudi L; Kahn B B
CS Department of Medicine at Harvard Medical School and Beth Israel Hospital, Boston, Massachusetts, USA.. jrz@klinfys.ks.se
NC RO1 DK-43051 (NIDDK)
SO DIABETES, (1997 Feb) 46 (2) 215-23.
Journal code: E8X. ISSN: 0012-1797.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199704
AB **Glucose** transport in skeletal muscle can be mediated by two separate pathways, one stimulated by insulin and the other by muscle contraction. High-fat feeding impairs **glucose** transport in muscle, but the mechanism remains unclear. FVB mice (3 weeks old) were fed a high-fat diet (55% fat, 24% carbohydrate, 21% protein) or standard chow for 3-4 weeks or 8 weeks. Insulin-stimulated **glucose** transport, assessed with either 2-deoxyglucose or 3-O-methylglucose was decreased 35-45% (P < 0.001) in isolated soleus muscle, regardless of diet duration. Similarly,

glucose transport stimulated by okadaic acid, a serine/threonine phosphatase inhibitor, was also 45% lower with high-fat feeding, but the **glucose** transport response to hypoxia or N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide (W-7) (which are stimulators of the "contraction pathway") was intact. **Hexokinase** I, II, and total activity were normal in soleus muscle from high-fat-fed mice. GLUT4 expression in soleus muscle from the high-fat-fed mice was also normal, but the insulin-stimulated cell surface recruitment of GLUT4 assessed by exofacial photolabeling with [3H]-ATB bis-mannose was reduced by 50% ($P < 0.001$). Insulin-receptor substrate 1 (IRS-1) associated phosphatidylinositol (PI) 3-kinase activity stimulated by insulin was also reduced by 36% ($P < 0.001$), and expression of p85 and p110b subunits of PI 3-kinase was normal. In conclusion, high-fat feeding selectively impairs insulin-stimulated, but not contraction-pathway-mediated, **glucose** transport by reducing GLUT4 translocation to the **plasma** membrane. This appears to result from an acquired defect in insulin activation of PI 3-kinase. Since effects of okadaic acid on **glucose** transport are independent of PI 3-kinase, a second signaling defect may also be induced.

L14 ANSWER 6 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3
AN 96:127207 BIOSIS
DN 98699342
TI **Compartment** analysis of cerebral **glucose** metabolism and in vitro **glucose**-metabolizing enzyme activities in the rat brain.
AU Ouchi Y; Fukuyama H; Matsuzaki S; Ogawa M; Kimura J; Tsukada H; Kakiuchi T; Kosugi T; Nishiyama S
CS Dep. Neurol., Fac. Med., Kyoto Univ., 54 Shogoin Kawahara-cho, Sakyo-ku, Kyoto, Japan
SO Brain Research 706 (2). 1996. 267-272. ISSN: 0006-8993
LA English
AB To clarify the relationship between cerebral **glucose** metabolic rate constants and **glucose**-metabolizing enzyme activities in the cerebral cortex, we evaluated the cerebral metabolic rate of **glucose** (CMRGlu), metabolic rate constants of (18F)-2-fluoro-2-deoxy-D-**glucose** (FDG) and related enzyme activities in the frontal cortex under normal and **glucose** metabolism-suppressed conditions. Applying a three-**compartment** four-parameter model, metabolic rate constants were obtained by dynamic positron emission tomography with FDG, and CMRGlu was calculated based on these rate constants. The glycolytic enzyme activities were determined by in vitro biochemical assay. Three days after ibotenic acid injection into the basal forebrain, CMRGlu was decreased in the ibotenic acid-treated frontal cortex as well as k3* (phosphorylation), while K1* (**plasma** to brain) showed no remarkable change. No significant reductions of the enzyme

activities except for **hexokinase** activity were found in the frontal cortex. Regression analysis showed a significant positive correlation between k3* and the **hexokinase** activity. These results suggested that k3* in the **compartment** analysis reflects **hexokinase** activity.

L14 ANSWER 7 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 4
AN 97:82475 BIOSIS
DN 99374188
TI Effects of pressure on whole **blood glucose** measurements using the Bayer Glucometer 4 **blood glucose** meter.
AU Edge C J; Grieve A P; Gibbins N; O'Sullivan F; Bryson P
CS Diving Dis. Res. Cent., Tamar Sci. Park, Derriford Road, Plymouth PL6 8BQ, UK
SO Undersea & Hyperbaric Medicine 23 (4). 1996. 221-224. ISSN: 1066-2936
LA English
AB The effect of pressure was investigated on the readings of whole **blood glucose** obtained from the Bayer Glucometer 4 **blood glucose** meter which uses the **hexokinase** enzymatic reaction. Sixteen subjects (eight normal and eight insulin-dependent diabetics) were exercised in a hyperbaric **chamber** at a depth of 3.7 atm abs. Venous **blood samples** were monitored at regular intervals for whole **blood glucose** concentration as measured by a Glucometer 4 inside the **chamber**. The **blood samples** were immediately placed in an airlock and taken to 1 atm abs, where whole **blood glucose** concentrations were measured using an identical instrument. The remaining **blood** was then analyzed in duplicate for serum **glucose** concentration using standard laboratory methods. The results show a significant difference between whole **blood glucose** concentrations measured at pressure and those measured at atmospheric pressure. Significant differences are also observed between whole **blood glucose** concentrations measured under pressure and serum **blood glucose** concentrations measured at atmospheric pressure.

L14 ANSWER 8 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 5
AN 96:271721 BIOSIS
DN 98827850
TI Hemocirculation and metabolism in intraventricular tumors: Kinetic analysis of **glucose** metabolism.
AU Shioya H; Mineura K; Kowada M; Iida H; Murakami M; Ogawa T; Hatazawa J; Uemura K
CS Neurosurgical Serv., Akita Univ. Hosp., 1-1-1 Hondo, Akita-city 010, Akita, Japan
SO Neurological Surgery 24 (3). 1996. 211-219. ISSN: 0301-2603

LA Japanese

AB To estimate hemocirculation and proliferating activity of intraventricular tumor, we measured kinetic rate constants (k1*, k2*, k3*) and **glucose** metabolic rate (kinetic-rCMRGl) using dynamic positron emission tomography (PET), as well as regional cerebral **blood** flow (rCBF), **blood** volume (rCBV), oxygen extraction fraction (rOEF), oxygen metabolic rate (rCMRO-2) and autoradiographic rCMRGl (arg-rCMRGl), in patients with intraventricular tumor. The subjects included ten patients, five males and five females, aged from 13 to 53 years with a mean age of 32 years old. Eight tumors were located in the lateral ventricle and two extended into the third ventricle through the foramen of Monro. Another two tumors were located in the fourth ventricle. Histological diagnosis was as follows : five cases of central neurocytoma, one subependymal giant cell astrocytoma, one ependymoma, one choroid plexus carcinoma, one subependymoma, and one meningioma. Tumor lesion on the PET images was determined using CT or MRI, which was performed at levels equivalent to those for the PET scans. For quantitative analysis, regions of interest (ROI) on PET images were delineated on the tumor and the contralateral gray matter. Hemocirculation (rCBF, rCBV) of the tumor was similar to or higher than that of the contralateral gray matter, which corresponded to neuroradiological findings of abundant tumor vessels. Oxygen metabolic parameters (rOEF, rCMRO-2) were significantly lower than those of the contralateral gray matter. Especially, low rOEF resulted in an excessive **blood** flow beyond oxygen demand of the tumor. The raised metabolic rate (rCMRO-2/rCMRGl), as compared with that of meningiomas or malignant gliomas, suggested aerobic glycolysis. The kinetic rate constants of tracer transport from **blood** to brain (k1*), reverse transport from brain to **blood** (k2*), and phosphorylation (k3*) were analyzed according to the three-**compartment** model of 18F-fluorodeoxyglucose (18FDG). Tumor k1* and k2* values were similar to or higher than those of the contralateral gray matter, suggesting high permeability due to lack of **blood**-brain barrier and an abundant **blood** supply. Tumor k3* value, an indicator of **hexokinase** activity, and kinetic-rCMRGl were lower in six of eight patients. These six patients have been free from tumor recurrence or regrowth, postoperatively. In the other two patients, tumor kinetic-rCMRGl was similar to or higher than that of the contralateral gray matter, suggesting high activity of proliferation. However, one patient received irradiation and has been followed up, and the other received total resection and has shown no recurrence. Functional information concerning intraventricular tumor is obtained by PET analysis, and kinetic analysis of the rate constants is useful for interpreting a detailed metabolic process of **glucose**, and provides additional information on intraventricular tumor aggressiveness.

AN 97:24181 BIOSIS
DN 99323384
TI **Glucose** uptake in Plasmodium falciparum-infected erythrocytes is an equilibrative not an active process.
AU Kirk K; Horner H A; Kirk J
CS Div. Biochem. Molecular Biol., Fac. Sci., Australian Natl. Univ., Canberra, A.C.T. 0200, Australia
SO Molecular and Biochemical Parasitology 82 (2). 1996. 195-205. ISSN: 0166-6851
LA English
AB The uptake of **glucose** into human erythrocytes infected with Plasmodium falciparum was investigated using a number of different **glucose** analogues. In short time-courses with cells suspended in media containing 5 mM **glucose**, 2-deoxy-D-**glucose** equilibrated rapidly between the intracellular and extracellular **compartments**. Its transport into the infected cell was primarily via the host cell (cytochalasin B-sensitive) transporter. 2-Deoxy-D-**glucose** did permeate the broad-specificity pathway that is induced in infected cells by the intracellular parasite. However, this pathway made little contribution to the total uptake of 2-deoxy-D-**glucose** under physiological conditions. In parasitised cells incubated with (14C)2-deoxy-D-**glucose** for prolonged periods the intracellular concentration of radiolabel increased to values higher than that in the external medium; it reached a maximum value three to six times higher than the extracellular concentration before falling back to a concentration similar to that outside the cells. This transient intracellular accumulation of radiolabel was due entirely to the phosphorylation of the (14C)2-deoxy-D-**glucose** and its consequent trapping within the cell. The specific characteristics of the 2-deoxy-D-**glucose** uptake time courses measured under different conditions were accounted for by the kinetics of the phosphorylation process and the energy status of the cell. The data indicate that 2-deoxy-D-**glucose** (as well as the non-phosphorylated compounds 3-O-methyl-D-**glucose** and L-**glucose**) enter the intracellular parasite via a passive (i.e. equilibrative) rather than an active (i.e. concentrative) transport process.

L14 ANSWER 10 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:409479 BIOSIS
DN 99131835
TI The metabolic organization of the adult human liver: A comparative study of normal, fibrotic, and cirrhotic liver tissue.
AU Racine-Samson L; Scoazec J-Y; D'Errico A; Fiorentino M; Christa L; Moreau A; Roda C; Grigioni W F; Feldmann G
CS Laboratoire de Biologie Cellulaire, Unite INSERM 327, Faculte de Medecine Xavier Bichat, BP 416, 75870 Paris Cedex 18, France
SO Hepatology 24 (1). 1996. 104-113. ISSN: 0270-9139
LA English

AB Little is known about the alterations of metabolic organization of the human liver tissue in chronic liver diseases. We therefore compared the distribution of the following zonal metabolic markers in 10 **samples** of normal liver tissue, 10 **samples** of fibrotic tissue, and 22 **samples** of cirrhotic tissue: (a) the enzymatic activities of **glucose**-6-phosphatase (G6P), lactate dehydrogenase (LDH), succinate dehydrogenase (SDH), nicotinamide-adeninedinucleotide-phosphate (NADPH) dehydrogenase (ND), beta-hydroxybutyrate dehydrogenase (HBDH), and glutamate dehydrogenase (GDH); (b) the protein glutamine synthetase (GLS); and (c) albumin messenger RNA (mRNA). The normal human hepatic lobule was characterized by the periportal predominance of G6P and SDH enzymatic activities and albumin mRNAs, the perivenous predominance of ND and GDH, the restriction of GLS to a small perivenous **compartment**, and the predominance of beta-HBDH at the contact of both portal tracts and centrilobular veins. In fibrosis, the overall metabolic organization of the normal liver tissue was retained. The expression of periportal markers predominated around enlarged portal tracts and that of perivenous markers around residual centrilobular veins. GLS was constantly detected at the contact of centrilobular veins. In cirrhotic nodules, no zonation was observed for most enzymatic activities or for albumin. Only G6P usually predominated at the periphery of the nodules. GLS was constantly undetectable. No difference according to the etiology of the underlying disease was observed. In conclusion, the normal human hepatic lobule presents a marked metabolic zonation, preserved in fibrotic lesions, but lost in cirrhotic nodules. The alterations of the metabolic organization observed in cirrhosis might contribute to the pathogenesis of some of the metabolic disorders associated with advanced liver disease.

L14 ANSWER 11 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 7
AN 96:55577 BIOSIS
DN 98627712
TI Net sugar transport is a multistep process. Evidence for cytosolic sugar binding sites in erythrocytes.
AU Cloherty E K; Sultzman L A; Zottola R J; Carruthers A
CS Dep. Biochem. Mol. Biol., Program Mol. Med., Univ. Mass. Med. Sch., Two Biotech, 373 Plantation St., Worcester, MA 01605, USA
SO Biochemistry 34 (47). 1995. 15395-15406. ISSN: 0006-2960
LA English
AB Human erythrocyte net sugar transport is hypothesized to be rate-limited by reduced cytosolic diffusion of sugars and/or by reversible sugar association with intracellular macromolecules (Naftalin, R. J., Smith, P. M., & Roselaar, S. E. (1985) Biochim. Biophys. Acta 820, 235-249). The present study examines these hypotheses. Protein-mediated 3-O-methylglucose uptake at 4 degree C by human erythrocytes and by resealed, hypotonically lysed erythrocytes (ghosts) is inhibited by increasing solvent viscosity. Protein-mediated transport and transbilayer diffusion of the slowly

transported substrate 6-NBD glucosamine are unaffected by increasing solvent viscosity. These findings suggest that protein-mediated 3-O-methylglucose transport is diffusion-limited in erythrocytes. More detailed analyses of red cell 3-O-methylglucose uptake (at 4 degree C and at limiting extracellular sugar levels) reveal that net influx is a biexponential process characterized by rapid filling of a small **compartment** (C-1 = 29 +- 6% total cell volume; k-1 = 7.4 +- 1.7 min-1) and slow filling of a larger **compartment** (C-2 = 71 +- 6% total cell volume; k-2 = 0.56 +- 0.11 min-1). Erythrocyte D-**glucose** net uptake at 4 degree C is also a biphasic process. Transmembrane sugar leakage is a monoexponential process indicating that multicomponent, protein-mediated uptake does not result from sugar uptake by two cell populations of differing cellular volume. Sugar exit at limiting 3-O-methylglucose concentrations is described by single exponential kinetics. This demonstrates that multicomponent sugar uptake does not result from influx into two populations of cells with widely different sugar transporter content. We conclude that biexponential sugar uptake results from slow (relative to transport) exchange of sugars between serial, intracellular sugar **compartments**. Biexponential sugar uptake is observed under equilibrium exchange conditions (intracellular sugar concentration = extracellular sugar concentration) but only at 3-O-methylglucose concentrations of less than 1 mM. Above this sugar concentration, exchange uptake is a monoexponential process. Because diffusion rates are independent of diffusant concentration, this suggests that multicomponent uptake results from high-affinity sugar binding within the cell. The concentration of cytosolic binding sites (30 mu-M, K-d(app) = 400 mu-M) was estimated from the equilibrium cellular 3-O-methylglucose space. Biexponential net 3-O-methylglucose uptake is also observed in human erythrocyte ghosts, in control human K562 cells, and in K562 cells induced to synthesize hemoglobin by prolonged exposure to hemin. This demonstrates that neither membrane-bound nor free cytosolic hemoglobin forms the sugar-binding complex. alpha-Toxin-permeabilized cells fill rapidly (within 5 s) with 3-O-methylglucose and L-**glucose** (a nontransported sugar), indicating that the **glucose** binding **compartment** does not extend across the entire intracellular margin of the **plasma** membrane. Rather, it must be restricted to domains of locally high-**glucose** transporter density. Immunofluorescence microscopy of erythrocytes indicates that GLUT1 is not distributed uniformly across the cell surface, while the anion transporter shows a uniform cell surface distribution. Red cell **hexokinase** I and GLUT1 appear not to colocalize in hypotonically lysed erythrocytes. The kinetics of sugar uptake and exit are quantitatively mimicked by a model in which newly imported sugars enter the bulk intracellular water only following interaction with an intracellular, sugar-binding complex. We conclude that steady state sugar transport assays in human erythrocytes measure two

processes: rapid sugar translocation across the bilayer and slow sugar release into bulk cytosol. The conclusions of previous steady state analyses which assume net transport reflects only sugar translocation may require reconsideration.

L14 ANSWER 12 OF 43 MEDLINE
AN 96125545 MEDLINE
DN 96125545
TI Lama glama (the South American camelid, llama): a unique model for evaluation of xenogenic islet transplants in a cerebral spinal **fluid** driven artificial organ.
AU Ommaya A K; Atwater I; Yanez A; Szpak-Glasman M; Bacher J; Arriaza C; Baer L; Parraguez V; Navia A; Oberti C; et al
CS Laboratory of Cell Biology and Genetics, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD, USA.
SO TRANSPLANTATION PROCEEDINGS, (1995 Dec) 27 (6) 3304-7.
Journal code: WE9. ISSN: 0041-1345.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199604

L14 ANSWER 13 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 8
AN 95:551453 BIOSIS
DN 98565753
TI Kinetics of **glucose** metabolism in central neurocytomas.
AU Shioya H; Mineura K; Sasajima T; Kowada M; Iida H; Ogawa T; Hatazawa J; Uemura K
CS Neurosurg. Serv., Akita Univ. Hosp., 1-1-1 Hondo, Akita 010, Japan
SO Brain and Nerve (Tokyo) 47 (10). 1995. 981-987. ISSN: 0006-8969
LA Japanese
AB To estimate proliferating activity of central neurocytoma, we measured kinetic rate constants and **glucose** metabolic rate (kinetic-rCMRGl) using dynamic positron emission tomography (PET), as well as autoradiographic rCMRGl (arg-rCMRGl), in patients with histologically verified central neurocytoma. The subject included five patients, four males and one female, aged from 23 to 53 years with a mean age of 41 years old. All tumors were located in the lateral ventricle and two extended into the third ventricle through the foramen of Monro. Tumor lesion on the PET images was determined using CT or MRI, which was performed at levels equivalent to those for the PET scans. The kinetic rate constants of tracer transport from **blood** to brain (k1), reverse transport from brain to **blood** (k2), and phosphorylation (k3) were analyzed according to the three **compartment** 18F-fluorodeoxyglucose (FDG) model. For quantitative analysis, regions of interest (ROI) on PET images were delineated on the tumor and the contralateral gray

matter. Tumor k1 and k2 values were similar to or higher than those of the contralateral gray matter, suggesting high permeability due to lack of **blood**-brain barrier. Tumor k3 value, an indicator of **hexokinase** activity, and kinetic-rCMRGl were exceedingly lower in three of five patients. These three patients have been free from tumor recurrence or regrowth, postoperatively. The other two patients, tumor kinetic-rCMRGl was similar to or higher than that of the contralateral gray matter. One patient suffered from tumor regrowth shortly after resection, and the other has been followed up postoperatively. Thus, k3 and kinetic-rCMRGl are indicative parameters of proliferative activity in central neurocytoma.

L14 ANSWER 14 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 9
AN 95:396250 BIOSIS
DN 98410550
TI Kinetic analysis of **glucose** metabolism in Meningiomas: Comparison with malignant gliomas.
AU Shioya H; Mineura K; Sasajima T; Kowada M; Iida H; Ogawa T; Hatazawa J; Oemura K
CS Neurosurgical Serv., Akita Univ. Hosp., 1-1-1 Hondo, Akita-city 010, Akita, Japan
SO Brain and Nerve (Tokyo) 47 (6). 1995. 549-556. ISSN: 0006-8969
LA Japanese
AB We measured kinetic rate constants and **glucose** metabolic rate (kinetic-rCMRG1) using dynamic positron emission tomography (PET), as well as regional cerebral **blood** flow (rCBF), **blood** volume (rCBV), oxygen extraction fraction (rOEF), oxygen metabolic rate (rCMRO-2) and autoradiographic rCMRG1 (arg-rCMRG1), in patients with meningioma. Ten patients including one recurrent case, two males and eight females aged from 44 to 71 years with a mean age of 54 years old, were studied prior to surgical interventions. Histological diagnosis was as follows: seven cases of meningothelial type, two cases of angiomatous type and one fibrous type. For quantitative analysis, regions of interest (ROI) on PET images were delineated on the tumor and the contralateral gray matter in comparison with eight cases with malignant gliomas (five cases of malignant astrocytoma and three cases of glioblastoma, aged from 14 to 70 years with a mean age of 41 years old). Hemocirculation of the tumor was exceedingly higher than that of the contralateral gray matter, which corresponded to neuroradiological findings of abundant tumor vessels. Low rOEF implicated an excessive **blood** flow beyond oxygen demand of the tumor. The raised metabolic rate (rCMRO-2/rCMRG1) suggested rather aerobic glycolysis as compared with malignant gliomas. The kinetic rate constants of tracer transport from **blood** to brain (k1), reverse transport from brain to **blood** (k2), and phosphorylation (k3) were analyzed according to the three **compartment** model of 18F-fluorodeoxyglucose (FDG). Tumor k1 and k2 values markedly increased in all examined cases, suggesting high permeability due to lack of **blood**

-brain barrier and an abundant **blood** supply. Tumor k3 value, an indicator of **hexokinase** activity, was lower than that of the contralateral gray matter in all cases but one. The remaining one patient showing an increased tumor k3 suffered from tumor recurrence within one year after extensive resection (Simpson's Grade IV). Thus, increased k3 may indicate high activity of proliferation. Kinetic analysis of the rate constants is useful for interpreting detailed metabolic process of **glucose**, and provides additional information on tumor aggressiveness.

L14 ANSWER 15 OF 43 JICST-EPlus COPYRIGHT 1998 JST
AN 960739911 JICST-EPlus
TI Kinetic Analysis of **Glucose** Metabolism in Angiomatous Meningioma.
AU SHIOYA HITOSHI; MINEURA KATSUYOSHI; SASAJIMA TOSHIO; KOWADA MASAYOSHI
IIDA HIDEHIRO; OGAWA TOSHIHIDE; HATAZAWA JUN; UEMURA KAZUO
CS Akita Univ.
Res. Inst. of Brain and Blood Vessel. Akita
SO CI Kenkyu (Progress in Computed Imaging), (1995) vol. 17, no. 3, pp. 293-298. Journal Code: G0734B (Fig. 5, Tbl. 2, Ref. 11)
ISSN: 0918-7073
CY Japan
DT Journal; Short Communication
LA Japanese
STA New
AB We measured the kinetic rate constants and **glucose** metabolic rate (kinetic rCMRG1) in an angiomatous meningioma by using dynamic positron emission tomography (PET). In addition, we also determined rCBF, rCBV, rOEF, rCMRO2 and autoradiographic rCMRG1. A 50-year-old woman who presented with gait disturbance and recent memory disturbance was admitted to our hospital. The MR images disclosed a mass lesion that occupied the right anterior fossa. Cerebral angiograms showed a sun-burst appearance via the right middle meningeal artery, and a tumor stain fed by the right orbitofrontal artery. The hemocirculation of the tumor was strikingly higher than that of the contralateral gray matter, corroborating the neuroradiological findings of abundant tumor vessels. The rate constants were determined according to the three **compartment** model of 18F-fluorodeoxyglucose (FDG). The tumor k1 and k2 values were markedly increased, which suggested a high permeability due to the lack of **blood** tumor barrier and abundant **blood** supply. On the other hand, the tumor k3 value, an indication of **hexokinase** activity was lower than that of the contralateral gray matter. The autoradiographic rCMRG1 was calculated by using the rate constants of normal brain tissue determined by Phelps et al. and the kinetic rCMRG1 was measured using the rate constants obtained from the dynamic analysis developed by Sasaki et al. Our data indicate that in the present

case the kinetic rCMRG1 was lower than the autoradiographic rCMRG1. After embolization of the feeding arteries, the patient's tumor was extirpated through a right frontotemporal craniotomy, the histological diagnosis was angiomatous meningioma. From our results it is evident that the kinetic analysis of the rate constants is useful for the detailed interpretation of the processes involved in the metabolism of **glucose** and for predicting biological tumor malignancy. (author abst.)

L14 ANSWER 16 OF 43 LIFESCI COPYRIGHT 1998 CSA
AN 96:26318 LIFESCI
TI **Compartment** analysis of cerebral **glucose** metabolism and in vitro **glucose**-metabolizing enzyme activities in the rat brain
AU Ouchi, Y.; Fukuyama, H.; Matsuzaki, S.; Ogawa, M.; Kimura, J.; Tsukada, H.; Kakiuchi, T.; Kosugi, T.; Nishiyama, S.
CS Department of Neurology, Faculty of Medicine, Kyoto University, 54 Shogoin Kawahara-cho, Sakyo-ku, Kyoto, Japan
SO BRAIN RES., (1995) vol. 706, no. 2, pp. 267-272.
ISSN: 0006-8993.
DT Journal
FS N3
LA English
SL English
AB To clarify the relationship between cerebral **glucose** metabolic rate constants and **glucose**-metabolizing enzyme activities in the cerebral cortex, we evaluated the cerebral metabolic rate of **glucose** (CMRGlu), metabolic rate constants of [18F]-2-fluoro-2-deoxy-d-**glucose** (FDG) and related enzyme activities in the frontal cortex under normal and **glucose** metabolism-suppressed conditions. Applying a three-**compartment** four-parameter model, metabolic rate constants were obtained by dynamic positron emission tomography with FDG, and CMRGlu was calculated based onthese rate constants. The glycolytic enzyme activities were determined by in vitro biochemical assay. Three days after ibotenic acid injection into the basal forebrain, CMRGlu was decreased in the ibotenic acid-treated frontal cortex as well as k3* (phosphorylation), while K1* (**plasma** to brain) showed no remarkable change. No significant reductions of the enzyme activities except for **hexokinase** activity were found in the frontal cortex. Regression analysis showed a significant positive correlation between k3* and the **hexokinase** activity. These results suggested that k3* in the **compartment** analysis reflects **hexokinase** activity.

L14 ANSWER 17 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 10
AN 95:80261 BIOSIS
DN 98094561
TI Longitudinal analysis of **glucose** metabolism in recurrent

meningioma.

AU Shioya H; Mineura K; Sasajima T; Kowada M; Iida H; Ogawa T; Hatazawa J; Uemura K

CS Neurosurg. Serv., Akita Univ. Hosp., 1-1-1 Hondo, Akita 010, Japan

SO Brain and Nerve (Tokyo) 46 (11). 1994. 1088-1093. ISSN: 0006-8969

LA Japanese

AB We repeatedly measured kinetic rate constants and **glucose** metabolic rate (kinetic rCMRGl) using dynamic positron emission tomography (PET) and autoradiographic rCMRGl in a patient with recurrent meningioma. A 50-year-old woman who presented with a left visual disturbance was admitted to our hospital. MR images revealed a mass lesion occupying the left middle fossa. The patient underwent Simpson grade IV surgery. The histological diagnosis was meningothelial meningioma. One year later the tumor had grown back to almost the same size as before treatment and was removed again by Simpson grade IV procedure. Postoperatively, the patient underwent radiation therapy (54 Gy). Two years after the second operation, the tumor was found to have invaded the left orbit and was resected by Simpson grade IV procedure. After additional radiation therapy, the patient was discharged. The rate constants were analyzed preoperatively and whenever the tumor recurred according to the three **compartment** 18F-fluorodeoxyglucose (FDG) model. Preoperative PET indicated tumor k1 and k2 values higher than in the contralateral gray matter, suggesting high permeability due to absence of the **blood**-tumor barrier and an abundant **blood** supply. The tumor k3 value, an indicator of **hexokinase** activity, was as high as in the contralateral gray matter. When the tumor recurred, the tumor k1, k2 and k3 values remained consistently high, indicating high proliferative activity. In contrast, the contralateral gray matter k1, k2 and k3 values decreased to some extent, suggesting effects of surgery or radiotherapy. Tumor rCMRGl values, both autoradiographic and kinetic, were enhanced markedly. Kinetic rate constants, such as k1, k2 and k3 values, provide additional information on **glucose** metabolism and are helpful in predicting tumor aggressiveness and the probability of recurrence.

L14 ANSWER 18 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.DUPLICATE 11

AN 94275692 EMBASE

TI Spatio-temporal regulation of glycolysis and oxidative phosphorylation in vivo in tumor and yeast cells.

AU Cortassa S.; Aon M.A.

CS Depto. de Bioquimica de la Nutricion, Instituto de Quimica Biologica, Universidad Nacional de Tucuman, Chacabuco 461, 4000 - San Miguel de Tucuman, Argentina

SO CELL BIOL. INT., (1994) 18/7 (687-713).
ISSN: 1065-6995 CODEN: CBIIEV

CY United Kingdom

DT Journal
FS 029 Clinical Biochemistry
LA English
SL English
AB Recent advances in the in vivo control and regulation of glycolysis and oxidative phosphorylation in yeast and tumor cells is revised. New insights are presented from old and new experimental data interpreted in the light of powerful new technologies (e.g. NMR, confocal microscopy) and quantitative techniques combined with mathematical modeling. Those new aspects are mainly concerned with the dynamical organization of glycolysis and oxidative phosphorylation which emerges from the multiple interactions between **compartments** and processes inside the cells. Those **compartments** may be of structural origin, e.g. **plasma** membrane defining the cell boundary, mitochondrial-cytoplasmic, or functional ones such as the alternative association-dissociation of enzymes to subcellular structures (e.g. mitochondria, cytoskeleton) with different kinetic properties in each state. A novel regulatory mechanism concerning polymerization-depolymerization of microtubular protein may add a new dimension to the in vivo physiological properties of cells. One main suggestion coming from the modulatory power of the polymeric status and concentration of cytoskeleton components is that it could function as an intracellular mechanism of synchronization between microscopic (local) to macroscopic (global) processes. How the cell 'mixes' or switches on or off those regulatory steps or effectors under different physiological and environmental conditions and for different genetic backgrounds, is a main avenue of systematic research for the future.

L14 ANSWER 19 OF 43 MEDLINE DUPLICATE 12
AN 95066938 MEDLINE
DN 95066938
TI **Hexokinase** kinetics in human skeletal muscle after hyperinsulinaemia, hyperglycaemia and hyperepinephrinaemia.
AU Katz A; Raz I
CS Department of Clinical Physiology, Karolinska Institute, Karolinska Hospital, Stockholm, Sweden.
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1994 Aug) 151 (4) 527-30. Journal code: 1U4. ISSN: 0001-6772.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199502
AB The effects of 120 min of euglycaemic hyperinsulinaemia (UH, approximately 5 mM; 40 mU m-2 min-1), UH plus adrenaline infusion (0.05 microgram kg-1 min-1), and hyperglycaemic normoinsulinaemia (26 mM) on **hexokinase** kinetics in human skeletal muscle

were examined. Biopsies were obtained from the quadriceps femoris muscle before and after each clamp. Total muscle **hexokinase** activity (HKt) (measured on a 2500 g supernatant) at a saturating level of the substrate **glucose** (1 mM) averaged 13 mmol kg dry wt-1 min-1 in the basal state and did not change significantly under any condition. Soluble **hexokinase** activity (HKs) (16,000 g supernatant) accounted for approximately 65% of HKt in the basal state, and this percentage was not significantly affected by any condition, suggesting that there was no major transfer of HK between cytosol and mitochondria. The activity of HKt and HKs was inhibited by **glucose** 1,6-bisphosphate (G-1,6-P2) in a concentration dependent manner in the basal state, and the sensitivity to G1,6-P2 inhibition was not altered by any condition. The activity of HKt and HKs in the presence of a subsaturating level of **glucose** (0.1 mM) accounted for approximately 70% of the activity at 1 mM **glucose**, and this percentage was not altered by any condition. These data suggest that under the present conditions alterations in the rates of whole body **glucose** disposal cannot be associated with alterations in HK distribution between cellular **compartments** nor its measured kinetics properties.

L14 ANSWER 20 OF 43 MEDLINE
AN 94355344 MEDLINE
DN 94355344
TI Function of the outer mitochondrial **compartment** in regulation of energy metabolism.
AU Brdiczka D
CS Faculty of Biology, University of Konstanz, Germany..
SO BIOCHIMICA ET BIOPHYSICA ACTA, (1994 Aug 30) 1187 (2) 264-9. Ref: 39
Journal code: A0W. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals; Cancer Journals
EM 199412
AB Electron microscopy showed the organization of several kinases at the mitochondrial surface as complexes between outer membrane (porin), kinase, and inner membrane (presumably adenine nucleotide translocator?). The complexes were enriched in the isolated contact site fraction. Interaction of porin with the kinases in vitro led to formation of tetramers of **hexokinase** and active creatine kinase. Kinetic analyses of mitochondria with intact outer **compartment** showed separate ATP/ADP exchange between kinases and oxidative phosphorylation. Considering these results, we postulate that the mitochondrial metabolism in intact cells is not

regulated by free ADP, but induced by substrates wf kinases such as **glucose** or creatine (Fig 1). Increased ATP turnover in muscle during contraction results in only a small change in the free ADP but causes a larger change of creatine because the equilibrium constant of the creatine kinase reaction at pH 7.2 favours ATP formation (ATP creatine/ADP phosphocreatine = 104.7) [38]. In addition, the level of phosphocreatine is roughly 10-times higher compared to ATP. Considering the higher concentration and the equilibrium constant, it can be calculated that a change of ADP between 40 and 70 microM results in creatine increasing from 8 to 12 mM. Thus creatine can be the signal that stimulates the mitochondrial metabolism transmitted by the mitochondrial creatine kinase [39]. Likewise, increased **blood glucose** in muscle at rest or in the liver stimulates the mitochondrial metabolism transmitted by the activity of bound **hexokinase** utilizing external ATP. The mitochondrial metabolism provides the UTP for glycogen synthesis through mitochondrial nucleoside-diphosphate kinase activity (Fig 1).

L14 ANSWER 21 OF 43 JICST-EPlus COPYRIGHT 1998 JST
AN 940374957 JICST-EPlus
TI Non-invasive analysis of brain function. Cerebral circulation and metabolism measurement using positron emission tomography.
AU KANNO IWAO
CS Res. Inst. of Brain and Blood Vessels, Akita Prefecture
SO Shinkei Kenkyu no Shinpo (Advances in Neurological Sciences), (1994) vol. 38, no. 2, pp. 256-264. Journal Code: Z0693A (Fig. 11, Ref. 21) ISSN: 0001-8724
CY Japan
DT Journal; Commentary
LA Japanese
STA New
AB PET measures quantitative in vivo function by means of positron-emitter labeled tracers and positron emission tomography. The tracer behaves based on biological function of the living organ. PET devices quantitatively assess the concentration of the positron emitter tracer in tissues. The input function of the tracer is measured through withdrawal of arterial **blood**. The data are analyzed to obtain the biological function by means of the model that has been constructed based on biochemical and physiological knowledge. The model usually consists of several **compartments** each of which can be treated as a uniform virtual space for tracer behavior. O-15 labeled tracers are one of the most important and basic tracers in the current PET. Cerebral **blood** flow(CBF) can be measured by means of inhalation of C15O2 or intravenous injection of H215O. Both behave as diffusible tracer of H215O in brain, and its steady-state condition or clearance kinetics will provide quantitative CBF based on two-**compartment** model. Oxygen extraction fraction(OEF) was

measured with15O2 inhalation. Because of a short half-life, 15O tracer has an advantage to apply the steady-state approach for CBF and oxygen metabolism(CMRO2), and the other advantage is repeatability of the study. The repeatability is an important feature to evaluate pathologic brain functions like circulation reserve, metabolic reserve, and responsibility to perturbations by specific drug effect. The repeatability is also strong advantage as a tool for the activation study. **Glucose** metabolism can be measured by 18F labeled fluorodeoxyglucose(FDG). The FDG transports **blood** brain barrier(BBB) like natural **glucose** and being phospholyzed by the **hexokinase**, but not being further metabolized unlike the **glucose**. (abridged author abst.)

L14 ANSWER 22 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 94061372 EMBASE
TI Rapid postabsorptive metabolism of nicotinic acid in rat small intestine may affect transport by metabolic trapping.
AU Stein J.; Daniel H.; Whang E.; Wenzel U.; Hahn A.; Rehner G.
CS Division of Gastroenterology, Department of Internal Medicine, Johann-Wolfgang-Goethe-University, Theodor-Stern-Kai 7, D-60596 Frankfurt, Germany, Federal Republic of
SO J. NUTR., (1994) 124/1 (61-66).
ISSN: 0022-3166 CODEN: JONUAI
CY United States
DT Journal
FS 029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
AB Postabsorptive metabolism of [3H]nicotinic acid by rat proximal jejunum was studied in vitro using the everted sac technique and in vivo by applying ligated loops. Metabolites were analyzed by HPLC with radiochemical detection. Within 60 min of incubation nicotinic acid was almost completely metabolized in vivo. Only 3.2% of the label could be detected in the original substrate nicotinic acid, whereas >90% of the radioactivity present in the gut wall was nicotinamide. Further in vitro experiments revealed that conversion was rapid; after 10 min of incubation >40% of the substrate was converted to other metabolites. From analysis of the serosal **fluid**, it was evident that only nicotinic acid and nicotinamide were transferred to the serosal **compartment**. Rapid conversion of nicotinic acid might affect the transport step itself by metabolic trapping, resulting in the maintenance of a substrate gradient necessary for passive absorption. In contrast, as examined by gradient centrifugation, the substrate in subcellular organelles is transferred only to a minor extent.

L14 ANSWER 23 OF 43 WPIDS COPYRIGHT 1998 DERWENT INFORMATION LTD

GA The Genuine Article (R) Number: ET177
TI **GLUCOSE**-UPTAKE BY TRYPANOSOMA-BRUCEI - RATE-LIMITING STEPS IN GLYCOLYSIS AND REGULATION OF THE GLYCOLYTIC FLUX
AU TERKUILE B H (Reprint); OPPERDOES F R
CS INT INST CELLULAR & MOLEC PATHOL, TROP DIS RES UNIT, AVE HIPPOCRATE 74, B-1200 BRUSSELS, BELGIUM (Reprint)
CYA BELGIUM
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1991) Vol. 266, No. 2, pp. 857-862.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB **Glucose** uptake and metabolism in the bloodstream form of the glycosome-containing protozoan parasite Trypanosoma brucei was measured using C-14-labeled **glucose** in combination with the silicone oil centrifugation technique in short term (5-60 s) incubations. **Glucose** rather than **glucose** analogues was used to study the interrelation between the uptake process and the subsequent metabolic steps. **Glucose** uptake over the **plasma** membrane occurred by facilitated diffusion, which limited the overall glycolytic rate at external **glucose** concentrations (glc(out)) below 5 mM. At higher glc(out) another step, either transport over the glycosomal membrane or phosphorylation by **hexokinase** became rate-limiting. Mathematical modeling assuming that **glucose** uptake occurs by facilitated diffusion followed by an enzymatic step accurately predicts the experimental data. As predicted by the model, the internal concentration of non-metabolized **glucose** remains low till glc(out) = 5 mM and increases at higher external concentrations. In contrast to **glucose**, glycerol entered the cell by simple diffusion. Externally supplied glycerol did not affect **glucose** metabolism but externally added **glucose** interfered with glycerol metabolism in a way that suggests that the rate-limiting step is at the level of glycerol kinase. Our observations suggest that the bloodstream form of T. brucei adapts its **glucose** transport in a way that gives maximum yield at minimum expense.

L14 ANSWER 25 OF 43 JICST-EPlus COPYRIGHT 1998 JST
AN 910551832 JICST-EPlus
TI Insulin, Diabetic Condition, & Exercise.
AU TOYODA MASATERU
CS Toho Univ., School of Medicine, Ohashi Hospital
SO Toho Igakkai Zasshi (Journal of the Medical Society of Toho University), (1991) vol. 38, no. 1, pp. 4-8. Journal Code: G0654A (Fig. 2, Tbl. 1, Ref. 3)
CODEN: TOIZAG; ISSN: 0040-8670

CY Japan
DT Journal; General Review
LA Japanese
STA New
AB All of the observed effects of insulin on tissues could be the result of facilitated delivery of energy to anabolic processes. Under diabetic conditions, catabolic processes are accelerated in insulin-sensitive tissues, i.e., fat, muscle, and liver, and hyperglycemia is induced. Elevation of **blood glucose** must to utilize energy contained in insulin-insensitive tissues, e.g., nervous cells, lens, renal tubular cell, and basement membrane, and this promotes the anabolic process. This anabolic response may be involved with diabetic complications. Exercise, like insulin, delivers energy to the anabolic processes in muscle cells, and induces hypertrophy. It may be that two **compartments**, one being Insulin-Mitochondria-**Hexokinase** and the other CPK-MK-Mitochondria, supply energy to the anabolic process. (author abst.)

L14 ANSWER 26 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 91013420 EMBASE
TI Plasmodium falciparum carbohydrate metabolism: A connection between host cell and parasite.
AU Roth E. Jr.
CS Division of Hematology, Albert Einstein College of Medicine and Montefiore Hospital, 111 East 210th Street, Bronx, NY 10467, United States
SO BLOOD CELLS, (1990) 16/2-3 (453-460).
ISSN: 0340-4684 CODEN: BLCEDD
CY United States
DT Journal
FS 004 Microbiology
025 Hematology
029 Clinical Biochemistry
LA English
AB Selected aspects of the metabolism of Plasmodium falciparum are reviewed, but conclusions based on the study of other species of plasmodia are intentionally not included since these may not be applicable. The parasites increase **glucose** consumption 50-100 fold as compared to uninfected red cells; most of the **glucose** is metabolized to lactic acid. The parasite contains a complete set of glycolytic enzymes. Some enzymes such as **hexokinase**, enolase and pyruvate kinase are vastly increased over corresponding levels in uninfected red cells. However, the pathway for synthesizing 2,3-diphosphoglycerate (2,3-DPG) is absent. Parasitized red cells show a decline in the concentration of 2,3-DPG which may function as an inhibitor for certain essential enzyme pathways. Pentose shunt activity is increased in absolute terms, but as a percent of total **glucose** consumption, there is a

decrease during parasite infection of the red cell. The parasite contains a gene for G6PD and can produce a small quantity of parasite-encoded enzyme. It is not clear if the production of this enzyme can be up-regulated in G6PD deficient host red cells. The NADPH normally produced by the pentose shunt can be obtained from other parasite pathways (such as glutamate dehydrogenase). NADPH may subserve additional needs in the infected red cell such as driving diribonucleotide reductase activity - a rate limiting enzyme in DNA synthesis. The role of NADPH in protecting the parasite-red cell system against oxidative stress (via glutathione reduction) remains controversial. Parasitized red cells contain about 10 times more NAD(H) than uninfected red cells, but the NADP(H) content is unchanged. Parasites can actively degrade host cell ATP; the purine moiety may be salvaged for parasite nucleic acid synthesis. Many further questions remain to be investigated. Of particular interest is the question of **compartments** and the permeability of these **compartments** in parasitized red cells.

L14 ANSWER 27 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 13
AN 90:200574 BIOSIS
DN BA89:107245
TI MICHAELIS-MENTEN CONSTRAINTS IMPROVED CEREBRAL **GLUCOSE** METABOLISM AND REGIONAL LUMPED CONSTANT MEASUREMENTS WITH FLUORINE-18 FLUORODEOXYGLUCOSE.
AU KUWABARA H; EVANS A C; GJEDDE A
CS POSITRON IMAGING LAB., MCCONNELL BRAIN IMAGING CENT., MONTREAL NEUROL. INST., 3801 UNIV. ST., MONTREAL, QUEBEC, CANADA H3A 2B4.
SO J CEREB BLOOD FLOW METAB 10 (2). 1990. 180-189. CODEN: JCBMDN ISSN: 0271-678X
LA English
AB In the three-**compartment** model of transfer of native **glucose** and [18F]fluorodeoxyglucose (FDG) into brain, both transport across the **blood**-brain barrier and phosphorylation by **hexokinase** can be described by the Michaelis-Menten equation. This permits the use of fixed transport (.tau. = K*1/K1) and phosphorylation (.phi. = k*3/k3) tracer and **glucose**. By substituting transfer constants of FDG for those of **glucose**, using .tau. and .pi.hi, the lumped constant was determined directly by positron tomography. The same constraints also eliminated k*2 and k*3 from the model, thus limiting the parameters to K* [equivalent to K*1k*3/(k*2 + k*3)], K*1, and the cerebral vascular volume (Vo). In six healthy elderly men (aged 61 .+-. 5 years), time-activity records of cerebral cortical regions were analyzed with .tau. = 1.1 and .pi.hi = 0.3. The results were compared with those of the conventional FDG method. At 20 min, the goodness of fit by the new equation was as good as that of the conventional method at 45 min. The estimates obtained by the constrained method had stable coefficients of variation. After 20 min, regional differences between the estimates were independent of time, although

we observed steady decreases of K* and (k*3). The decrease strongly suggested dephosphorylation of FDG-6-phosphate, particularly after 20 min. All estimates of variables with the constrained method were more accurate than those of the conventional method, including the cerebral **glucose** metabolic rate itself, as well as physiologically more meaningful, particularly with respect to k*2 and k*3.

L14 ANSWER 28 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 14
AN 89:246366 BIOSIS
DN BA87:127431
TI **BLOOD GLUCOSE** PARTITION AND LEVELS OF GLYCOLYTIC ENZYMES IN ERYTHROCYTES AND SOMATIC TISSUES OF PENGUINS.
AU ROSA R; RODRIGUES E; BACILA M
CS CIENCIAS AGRARIAS, UNIV. FEDERAL DO PARANA, CAIXA POSTAL 672-CURITIBA, PARANA, BRAZIL-80,000.
SO COMP BIOCHEM PHYSIOL B COMP BIOCHEM 92 (2). 1989. 307-312. CODEN: CBPBB8 ISSN: 0305-0491
LA English
AB 1. A comparative study was carried out on **blood glucose** partition and **glucose** metabolism of penguin erythrocytes and somatic tissues. Pygoscelidae penguins (Pygoscelis antarctica and P. papua) were used in these experiments. 2. **Blood glucose** partition was established by assaying whole **blood** and **plasma glucose** in several individuals of the gentoo and chinstrap penguins. 3. It was found that almost all the whole **blood** sugar is **compartmentalized** at the **plasma** site, the red **blood** cells being ineffective in regard to **glucose** metabolism. 4. Levels of **hexokinase**, phosphoglucose isomerase, phosphofructokinase, fructose bisphosphate aldolase, glyceraldehyde phosphate dehydrogenase, phosphoglycerate kinase, phosphopyruvate hydratase (enolase), pyruvate kinase, .alpha.-glycerolphosphate dehydrogenase and fructose bisphosphate phosphatase were estimated in the erythrocytes of both gentoo and chinstrap penguins, the same determinations being carried out also on the somatic tissues (leg muscle, breast muscle, heart muscle, liver and brain) of the gentoo.

L14 ANSWER 29 OF 43 BIOTECHDS COPYRIGHT 1998 DERWENT INFORMATION LTD
AN 86-08259 BIOTECHDS
TI Detection of ATP and creatine-kinase using an enzyme electrode; with immobilized **glucose**-oxidase; potential application as a biosensor
AU Davis G; Green M J; Hill H A O
LO University of Newcastle upon Tyne, Department of Clinical Biochemistry, The Medical School, Framlington Place, Newcastle upon Tyne NE2 4HH, U.K.
SO Enzyme Microb.Technol.; (1986) 8, 6, 349-52

CODEN: EMTED2
DT Journal
LA English
AN 86-08259 BIOTECHDS
AB An electrochemical method is described for the detection of ATP and creatine-kinase (EC-2.7.3.2) coupled through **hexokinase** (EC-2.7.1.1) to an amperometric **glucose** enzyme electrode. The **glucose** enzyme electrode contains immobilized **glucose**-oxidase (EC-1.1.3.4) from Aspergillus niger. The potentiostatically controlled steady state current measurements with the electrode are performed in a cell, with separate **compartments** for the reference and counter electrodes, at 37 deg. The assay could be used monitor creatine-kinase activity over the range 0.01-10 U/ml in biological **samples**. This system should be of use in the development of a single use biosensor for use in diagnosis. (17 ref)

L14 ANSWER 30 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 15
AN 85:308381 BIOSIS
DN BA79:88377
TI METABOLIC FLUXES BETWEEN CARBON-14-LABELED 2 DEOXY-D-**GLUCOSE** AND CARBON-14-LABELED 2 DEOXY-D-**GLUCOSE** 6-PHOSPHATE IN BRAIN IN-VIVO.
AU HUANG M-T; VEECH R L
CS LAB. METABOLISM, NIAAA, 12501 WASHINGTON AVE., ROCKVILLE, MD 20852.
SO J NEUROCHEM 44 (2). 1985. 567-573. CODEN: JONRA9 ISSN: 0022-3042
LA English
AB The rates of the phosphorylation and dephosphorylation of 2-deoxyglucose were measured in rat brain in vivo using tracer kinetic techniques. The rate constant for each reaction was estimated from 2 separate experiments with different protocols for tracer administration. Tracer amounts of [1-14C]2-deoxyglucose (1 .mu.Ci) were injected through the internal carotid artery (intraarterial experiment), or through the atrium (i.v.experiment). Brains were **sampled** by freeze-blowing at various times after the injection. In the intraarterial experiment, the rate constant for the forward reaction from 2-deoxyglucose to 2-deoxyglucose phosphate was calculated by dividing the initial rate of 2-deoxyglucose phosphate production by the 2-deoxyglucose content in brain. The rate constant for the reverse reaction from 2-deoxyglucose was calculated from the decay constant of 2-deoxyglucose phosphate. The rate constants estimated were 10.1 .+-. 1.4%/min (SD) and 3.00 .+-. 0.01%/min (SD), respectively, for the forward and reverse reactions. In the i.v. experiment, rate constants for both reactions were estimated by **compartmental** analysis. By fitting data to program SAAM-27, the rate constants for the forward and reverse reactions were estimated as 11.4 .+-. 0.4%/min (SD) and 5.1 .+-. 0.4%/min (SD), respectively. The rate constants determined were compared to those for the reactions between **glucose** and G-6-P, estimated

previously from labeled **glucoses**. The rate of **glucose** utilization measured by the 2-deoxyglucose method reflects the rate of the **hexokinase** reaction and not the rate of **glucose** utilization or brain energy utilization.

L14 ANSWER 31 OF 43 MEDLINE
AN 85175448 MEDLINE
DN 85175448
TI [Ultrastructural and enzyme chemical changes in dog testicles as affected by temporary interruption of **blood** circulation and use of cardiac massage].
Ul'trastrukturnye i fermentokhimicheskie izmeneniia iachek sobak v usloviiakh vremennogo prekrashcheniia krovoobrashcheniia i primeneniia kardiomassazhera.
AU Lysenko A I; Kirpatovskii I D; Glushakov A S; Perekrest P V
SO BIULLETEN EKSPERIMENTALNOI BIOLOGII I MEDITSINY, (1985 Mar) 99 (3) 376-9.
Journal code: A74. ISSN: 0365-9615.
CY USSR
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals; Cancer Journals
EM 198508
AB Slight ultrastructural and metabolic changes in the spermatogenic epithelium of the testicles were identified 10 min after cardiac arrest. One hour after direct mechanical cardiomassage (DMCM) a moderately pronounced edema of the intercellular spaces in the basal **compartment** of the seminiferous epithelium, normal content of lactate and succinate dehydrogenases, and a certain decrease in the activity of **glucose**-6-phosphate dehydrogenases and NAD- and NADP-**diaphorases** were noted. Preservation of tight junctions between Sertoli cells, presenting an impermeable testicular barrier, both after cardiac arrest and DMCM suggest that the hypoxic alterations in the structure and metabolism do not interfere with the use of the testicles for transplantation.

L14 ANSWER 32 OF 43 MEDLINE
AN 86047796 MEDLINE
DN 86047796
TI Characteristics of **hexokinase**, pyruvate kinase, and **glucose**-6-phosphate dehydrogenase during adult and neonatal reticulocyte maturation.
AU Jansen G; Koenderman L; Rijksen G; Cats B P; Staal G E
SO AMERICAN JOURNAL OF HEMATOLOGY, (1985 Nov) 20 (3) 203-15.
Journal code: 3H4. ISSN: 0361-8609.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals

EM 198602
AB Erythrocytes from adults and newborn infants (at term and premature) were separated by Percoll density gradient centrifugation into four fractions of increasing density. Glycolytic enzymes, especially the age-dependent ones, **hexokinase** (EC 2.7.1.1, HK), pyruvate kinase (EC 2.7.1.40, PK), and **glucose**-6-phosphate dehydrogenase (EC 1.1.1.49, G6PD) were studied during reticulocyte maturation and further red cell senescence. Analysis of the fraction with lowest density showed an almost linear and steep decline of HK, PK, and G6PD activity with a decreasing number of reticulocytes. In the next three fractions of increasing density, the activity decline was far less. These data are therefore illustrative for a biphasic activity decay pattern of HK, PK, and G6PD during both adult and neonatal red cell aging. The strong decline in HK activity could not be ascribed to the disappearance of a particulate (mitochondrial) bound fraction of the enzyme during reticulocyte maturation. All **hexokinase** activity in human reticulocytes was found to be cytosolic in contrast with rabbit reticulocytes in which 70% of HK activity was particulate.

L14 ANSWER 33 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 16
AN 85:229067 BIOSIS
DN BA79:9063
TI INTRACELLULAR LOCALIZATION OF RAT KIDNEY **HEXOKINASE** EVIDENCE FOR AN ASSOCIATION WITH LOW DENSITY MITOCHONDRIA.
AU PARRY D M; PEDERSEN P L
CS LAB. MOLECULAR CELLULAR BIOENERGETICS, DEP. PHYSIOLOGICAL CHEM., JOHNS HOPKINS UNIV., SCH. MED., BALTIMORE, MD. 21205.
SO J BIOL CHEM 259 (14). 1984. 8917-8923. CODEN: JBCHA3 ISSN: 0021-9258
LA English
AB The subcellular location of **hexokinase** was investigated in rat kidney. Both soluble and particulate locations are indicated by differential centrifugation. The particulate form is predominant, representing .apprx. 80% of the total activity. None of the activity is latent. Density gradient centrifugation followed by marker enzyme analysis reveals the presence of 2 populations of mitochondria with distinct densities. **Hexokinase** is associated primarily with the mitochondrial population having the lowere density. Association of **hexokinase** with brush border, **plasma** membrane, lysosomes and endoplasmic reticulum is considered unlikely on the basis of density gradient centrifugation and enzyme analysis. About 95% of the **hexokinase** activity associated with the mitochondrial fraction can be released in soluble form by repeated incubations with **glucose** 6-phosphate. An incubation time of .apprx. 4 min at 30.degree. C is required to achieve a maximal solubilizing effect. Release is accomplished without disrupting the mitochondrial **compartments**. **Hexokinase** is released also by treatment of the mitochondrial fraction with increasing concentrations of digitonin. This technique disrupts and

differentially releases the mitochondrial **compartments**. As observed with liver, but in contrast to that observed with tumor, the release of **hexokinase** from the mitochondrial fraction of kidney does not correlate with the release of enzymes known to mark the mitochondrial membranes or **compartments**. The 1st critical evidence about the subcellular location of **hexokinase** in kidney was provided. In this tissue **hexokinase** is associated primarily with low density mitochondria, a finding that adds credibility to the existence of this decrete population of mitochondria in vivo. This association of **hexokinase** with kidney mitochondria appears unique in that its release on submitochondrial fractionation does not correlate with the release of known mitochondrial marker enzymes. These results are directly relevant to those cells in the kidney which utilize **glucose** as an energy source. Evidently, the enhanced glycolytic capacity of these cells may be due, at least in part, to an association of **hexokinase** with low density mitochondria.

L14 ANSWER 34 OF 43 MEDLINE
AN 85023462 MEDLINE
DN 85023462
TI The relationship between **glucose** concentration and rate of lactate production by human erythrocytes in an open perfusion system.
AU Kuchel P W; Chapman B E; Lovric V A; Raftos J E; Stewart I M; Thorburn D R
SO BIOCHIMICA ET BIOPHYSICA ACTA, (1984 Oct 12) 805 (2) 191-203. Journal code: A0W. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 198502
AB A thermodynamically open system, based on an assembly of capillaries with semi-permeable walls was constructed in order to study glycolysis in human erythrocytes in high haematocrit suspensions. A phenomenological expression for the rate of lactate production as a function of **glucose** concentration was obtained. The rate was measured under steady-state conditions with low substrate concentrations (approx. 50 mumol/l). In a corresponding closed system, this concentration of **glucose** would be exhausted within a few minutes. A mathematical model of the whole system consisted of five differential equations, and involved parameters relating to flow rates, volumes of reaction **chambers**, the rates of lactate efflux from erythrocytes and the expression for the rate of lactate production by red cells. The binding of [14C]pyruvate to haemoglobin and the rate of efflux of [14C]lactate from red cells were measured to yield additional information for the model. The concentrations of ATP and 2,3-bisphosphoglycerate were

measured during the perfusion experiments, and a detailed analysis of a model of red cell **hexokinase** was carried out; the former two compounds inhibit **hexokinase** and alter the apparent Km and Vmax for **glucose** in vivo. These steady-state parameters were similar to the **glucose** concentration at the half-maximal rate of lactate production and the maximal rate, respectively. These findings are consistent with the known high control-strength for **hexokinase** in glycolysis in human red cells. The practical and theoretical validation of this perfusion system indicates that it will be valuable for NMR-based studies of red cell metabolism using a flow-cell in the spectrometer.

L14 ANSWER 35 OF 43 DISSABS COPYRIGHT 1998 UMI Company
AN 83:18159 DISSABS Order Number: AAR8328538
TI CHARACTERIZATION AND RECONSTITUTION OF MONOSACCHARIDE TRANSPORT SYSTEMS IN SACCHAROMYCES CEREVISIAE
AU FRANZUSOFF, ALEXIS JACOB [PH.D.]
CS STATE UNIVERSITY OF NEW YORK AT STONY BROOK (0771)
SO Dissertation Abstracts International, (1983) Vol. 44, No. 8B, p. 2308. Order No.: AAR8328538. 224 pages.
DT Dissertation
FS DAI
LA English
AB **Glucose** transport in baker's yeast exhibits high affinity and low affinity components. The low affinity component is known to be mediated by a ficilitated diffusion carrier and the high affinity component is known to be dependent on the presence of the cognate sugar kinases, **hexokinases** or glucokinase. Conflicting hypotheses exist as to (1) the mechanism of the high affinity process (i.e., whether or not it represents a vectorial phosphorylation process) and (2) the role of the carrier in the high affinity process (i.e., whether the same carrier participates in both processes in two different affinity states mediated directly by its interaction with the kinases or indirectly by its interaction with sugar metabolites).

Pool labelling experiments with kinase positive yeast were consistent with a vectorial phosphorylation process. Using mutant strains of yeast with one functionally operating species of each of the cognate sugar kinases showed that the kinase-mediated **glucose** uptake is not kinase-specific. Caution is urged in the interpretation of these results since difficulties of metabolism and **compartmentation** of sugars in whole cells of baker's yeast hampers unambiguous interpretation of the role of phosphorylation in sugar uptake.

Hybrid liposome-**plasma** membrane vesicles and reconstituted proteoliposomes were successfully prepared (from baker's yeast) which carry out stereospecific carrier-mediated, facilitated diffusion of D-**glucose**, including stimulated

equilibrium exchange and influx countertransport. Some properties and characteristics of these monosaccharide transport systems isolated from the whole cells are described, and some initial applications to study the mechanism of sugar uptake were performed with these systems. Galactose uptake with transport systems isolated from induced baker's yeast is described, as well as the report of a specific inhibitor of monosaccharide transport in S. cerevisiae.

L14 ANSWER 36 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 17
AN 84:316120 BIOSIS
DN BA78:52600
TI ATP ADP DEPENDENT PHOSPHORYLATIONS OF GLYCOLYSIS METABOLITES CREATINE AND GLYCEROL THEIR **COMPARTITION** AND THERMODYNAMIC RELATIONSHIP IN GASTROCNEMIUS MUSCLE CELL OF EXERCISE GUINEA-PIGS.
AU FERAUDI M; KOLB J; HASSEL M; WEICKER H
CS RUPRECHT-KARLS-UNIV. HEIDELBERG, MEDIZINISCHE POLIKLIN., ABT. PATHOPHYSIOL. SPORTMED., HOSPITALSTR. 3, D-6900 HEIDELBERG 1.
SO ARCH INT PHYSIOL BIOCHIM 91 (4). 1983 (RECD. 1984). 351-360. CODEN: AIPBAY ISSN: 0003-9799
LA English
AB The concentrations of following metabolites were determined in freeze-clamped gastrocnemius muscle **samples**: **glucose** 1-phosphate, **glucose** 6-phosphate, **glucose**, fructose 1,6-diphosphate, fructose 6-phosphate, D-glyceraldehyde 3-phosphate, dihydroxyacetone phosphate, phosphoenolpyruvate, pyruvate, glycerol 3-phosphate, glycerol, creatine phosphate, creatine, glycerate 3-phosphate, glycerate 2-phosphate, AMP, ADP, ATP and Pi. Within the limits of experimental error, concentration homeostatis for these metabolites is based at least in some cases on equilibria between enzymic transformations. Discrepancies between constant mass ratios measured and equilibrium constants allow the free energy variation .DELTA.G to keep creatine phosphate at a sufficiently high concentration to be calculated. For the phosphoglycerate mutase system, the equilibrium constant in controls and trained animals is unchanged and corresponds to that in vitro. Training hindered glycolysis and favored phosphorylation of creatine by glycerol 3-phosphate. Metabolites of the pyruvate kinase and **hexokinase** system cannot be homogeneously distributed in 1 space. The creatine kinase system is also separated from the **hexokinase** and pyruvate kinase system. A **compartition** of glycolytic process in gastrocnemius muscle seems to be inferred from these results.

L14 ANSWER 37 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 18
AN 83:235565 BIOSIS
DN BA75:85565
TI GENERALIZED **HEXO KINASE** EC-2.7.1.1 DEFICIENCY IN THE **BLOOD** CELLS OF A PATIENT WITH NONSPHEROCYTIC HEMOLYTIC ANEMIA.

AU RIJKSEN G; AKKERMAN J W N; VAN DEN WALL BAKE A W L; HOFSTEDE D P; STAAL G E J
CS DEP. HAEMATOL., DIV. MED. ENZYMOL., STATE UNIV. HOSP., 3500 CG UTRECHT, NETHERLANDS.
SO BLOOD 61 (1). 1983. 12-18. CODEN: BLOOAW ISSN: 0006-4971
LA English
AB In a patient with nonspherocytic hemolytic anemia, a **hexokinase** deficiency was detected in the red cells (residual activity .apprx. 25% of normal) and in **blood** platelets (20-35% of normal activity). Although the total **hexokinase** activity in lymphocytes was normal, the amount of **hexokinase** type I was decreased to .apprx. 50% of normal. The deficiency was compensated for by the appearance of type III **hexokinase**. **Compartmentation** studies with controlled digitonin-induced cell lysis showed that this type III enzyme was localized in the cytosol, while almost all **hexokinase** activity in normal lymphocytes is particulate. No abnormal lymphocyte functions could be detected. The patient was homozygous for the defect. The parents and 3 of 5 siblings of the patient were apparently heterozygous with residual activities of 50-67% of normal in their red cells but did not show any clinical signs of **hexokinase** deficiency. The variant enzyme had a slightly decreased affinity for MgATP2- and a strongly increased inhibition constant for **glucose** -1,6-diphosphate. Affinity for **glucose**, heat stability and pH optimum were normal. In the electrophoretic pattern of red cell **hexokinase**, only 1 subtype of **hexokinase** I could be detected, while in normal red cells at least 3 subtypes are present. In the heterozygous individuals, no enzymatic abnormalities could be detected, except for an aberration in the electropherogram of 1 sibling.

L14 ANSWER 38 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 19
AN 83:296976 BIOSIS
DN BA76:54468
TI **COMPARTMENTATION** OF **HEXO KINASE** EC-2.7.1.1 IN HUMAN **BLOOD** CELLS CHARACTERIZATION OF SOLUBLE AND PARTICULATE ENZYMES.
AU RIJKSEN G; STAAL G E J; BEKS P J; STREEFKERK M; AKKERMAN J W N
CS DEP. HAEMATOL., DIV. MED. ENZYMOL., STATE UNIV. HOSP., CATHARIJNENSINGEL 101, 3500 CG, UTRECHT, NETH.
SO BIOCHIM BIOPHYS ACTA 719 (3). 1982. 431-437. CODEN: BBACAQ ISSN: 0006-3002
LA English
AB The isozyme distribution, kinetic properties and intracellular localization of **hexokinase** (ADP:D-hexose-6-phosphotransferase, EC 2.7.1.1) were studied in erythrocytes, **blood** platelets, lymphocytes and granulocytes. Soluble and particulate fractions were separated by a rapid density centrifugation method after controlled digitonin-induced cell lysis.

In lymphocytes and platelets the major part of total activity was particle-bound (78 and 88%, respectively). In granulocytes and erythrocytes most of the **hexokinase** activity was found in the cytosol. All cell types, except granulocytes, contain mainly the type I isozyme. Platelets contain only type I **hexokinase**, while in lymphocytes a minor amount of type III is present in the soluble fraction (< 10% of total activity). The major constituent of granulocytes is type III **hexokinase** (70-80% of total activity), the remaining 20-30% is type I **hexokinase**. Erythrocytes contain a multibanded type I **hexokinase**. The substrate affinities of the type I **hexokinase** do not differ significantly between the different cell types or between soluble, bound and solubilized fractions. Only soluble **hexokinase** from lymphocytes shows a slightly decreased Km apparent for **glucose** ($P < 0.05$).

L14 ANSWER 39 OF 43 BIOSIS COPYRIGHT 1998 BIOSIS
AN 82:263478 BIOSIS
DN BA74:35958
TI EFFECT OF STORAGE AT MINUS 80 CELSIUS ON THE ACTIVITIES OF CYTOPLASMIC MITOCHONDRIAL AND MICROSOMAL ENZYMES IN RAT LIVER.
AU BODE C; MEINEL A
CS ZENTRUM INNERE MED., UNIV., MANNKOPFFSTR. 1, D-3550 MARBURG/LAHN.
SO J CLIN CHEM CLIN BIOCHEM 20 (1). 1982. 9-14. CODEN: JCCBDT ISSN: 0340-076X
LA English
AB The effect of storage -80.degree. C for 1-28 days on the activity of 12 enzymes [glucokinase, **glucose**-6-phosphate dehydrogenase, fructose-bisphosphatase, 6-phosphofructokinase, glutamate dehydrogenase, L-aspartate: 2-oxoglutrate aminotransferase, L-alanine: 2-oxo-glutarate aminotransferase, xanthine oxidase, ornithine carbamoyl transferase, **glucose**-6-phosphatase, citrate synthase and arginase] in intact liver tissue, liver extract and isolated hepatic microsomes was investigated. To find optimal conditions for tissue homogenization for this study, the effects of 3 types of homogenization on the activity of 10 enzymes [glutamate dehydrogenase, citrate synthase, L-aspartate: 2-oxoglutarate aminotransferase, L-alanine: 2-oxoglutarate aminotransferase, **glucose**-6-phosphate dehydrogenase, fructose-6-phosphate-kinase, **hexokinase**, glucokinase, D-fructose-bis-phosphatase, ornithine carbamoyl transferase] from different cell **compartments** were compared. The activities of glucokinase and phosphofructokinase decreased markedly during storage of both supernatant and liver tissue. Storage of liver tissue increased the activity of mitochondrial enzymes or isoenzymes. While this effect can be explained by additional disintegration of liver tissue caused by freezing and thawing for enzymes like glutamate dehydrogenase, other mechanisms may be involved in the prolonged increase observed in the activity of citrate synthase and xanthine oxidase storage. The

activity of a number of enzymes from the cytosol, mitochondria and microsomes decreased more markedly in the stored liver **samples** than in the stored supernatant or in the stored microsomal pellet.

L14 ANSWER 40 OF 43 DISSABS COPYRIGHT 1998 UMI Company
AN 81:26655 DISSABS Order Number: AAR8123923
TI THE ROLE OF MEMBRANE-BOUND GLYCOLYTIC ENZYMES IN SUPPLYING ATP TO THE NA-K PUMP IN HUMAN ERYTHROCYTES
AU MERCER, ROBERT WILLIAM [PH.D.]
CS SYRACUSE UNIVERSITY (0659)
SO Dissertation Abstracts International, (1981) Vol. 42, No. 5B, p. 1720. Order No.: AAR8123923. 75 pages.
DT Dissertation
FS DAI
LA English
AB The mature human red **blood** cell synthesizes ATP exclusively through glycolysis. In these cells it has been suggested that two enzymes of the glycolytic pathway, glyceraldehyde-3-phosphate dehydrogenase (GAPDH) and phosphogly-cerate kinase (PGK) are associated with the Na-K pump. GADPH in the presence of orthophosphate and NAD, converts glyceraldehyde-3-phosphate to 1,3 diphosphoglycerate (1,3-DPG). The subsequent enzyme in the pathway, PGK, catalyses the transfer of a phosphate group from 1,3-DPG to ADP forming ATP. It has been suggested that the ATP synthesized by membrane-bound GAPDH and PGK is **compartmentalized** in a "membrane pool" which is used preferentially by the Na-K pump. In human red cells, it was found that reduction of intracellular orthophosphate inhibits NA and K transport through the Na-K pump. To investigate the role of phosphate in the inhibition of the Na-K pump, inside out vesicles (IOVs) from red cells were prepared. With IOVs it was found that orthophosphate had little direct effect on Na transport through the pump. However, orthophosphate in the presence of other GAPDH-PGK substrates could activate Na transport through the pump. The ATP formed by the GAPDH-PGK reaction must be **compartmentalized** since the addition of **hexokinase** + **glucose**, which rapidly hydrolyzes ATP in the medium, had no effect on the stimulation of Na transport. The presence and size of the membrane pool of ATP was estimated using ('32)P-labeled orthophosphate. These results suggest that the GAPDH-PGK reaction is important in supplying ATP to the pump and that the ATP formed is **compartmentalized** in the membrane.

L14 ANSWER 41 OF 43 MEDLINE DUPLICATE 20
AN 82077060 MEDLINE
DN 82077060
TI Membrane-bound ATP fuels the Na/K pump. Studies on membrane-bound glycolytic enzymes on inside-out vesicles from human red cell membranes.

AU Mercer R W; Dunham P B
NC CA 19064 (NCI)
AM 27851 (NIADDK)
SO JOURNAL OF GENERAL PHYSIOLOGY, (1981 Nov) 78 (5) 547-68.
Journal code: I8N. ISSN: 0022-1295.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198204
AB ATP stimulates Na transport into inside-out vesicles (IOVs) made from human red cell membranes; strophanthidin inhibits the ATP-stimulated transport. The substrates for glyceraldehyde-3-phosphate dehydrogenase (GAPDH) and phosphoglycerate kinase (PGK) (glycolytic enzymes bound to the cytoplasmic surface of the red cell membrane) also stimulate Na transport into IOVs without added ATP. The elution of GAPDH from the membranes prevents the stimulation by the substrates, but not by exogenous ATP. **Hexokinase** plus **glucose** (agents that promote breakdown of ATP) prevent stimulation of Na transport by exogenous ATP but not by the substrates for GAPDH and PGK. [32P]orthophosphate is incorporated into a membrane-bound organic phosphate compound shown chromatographically to be ATP. The level of membrane-bound ATP is decreased when Na is added, and this decrease is inhibited by strophanthidin. When further synthesis of [32P]ATP is blocked by the addition of unlabeled orthophosphate, all of the membrane-bound [32P]ATP is dissipated by the addition of Na. From these observations it was concluded that membrane-bound glycolytic enzymes synthesize ATP and deposit it in a membrane-associated **compartment** from which it is used by the Na/K pump.

L14 ANSWER 42 OF 43 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 75025238 EMBASE
TI [Enzymatic **blood glucose** determination by means of the **chamber** paper analyzing technique: a screening method].
ENZYMATISCHE BLUTGLUKOSEBESTIMMUNG MIT DER KAMMER PAPIER ANALYSE TECHNIK (KAPA): EINE SCREENINGMETHODE.
AU Petermann B.; Thielmann K.; Horn A.; Hoppe H.
CS Inst. Physiol. Chem., Friedrich Schiller Univ., Jena, German Democratic Republic
SO DTSCH.GESUNDH.-WES., (1974) 29/18 (850-853).
CODEN: DEGEA3
LA German
AB An enzymatic **glucose** determination in the serum is presented by means of the **chamber** paper analysis technique on the basis of the **hexokinase**/D **glucose** 6 phosphate dehydrogenase reaction. The precision from day to day for sera from 82-258 mg **glucose**/100 ml serum ranged from 3.5

to 1.4% CV with daily double determinations corresponding to 4.9-2.0% CV for single determinations. As compared to the conventional optical test, there resulted a correlation coefficient of 0.99. For the detection of a **glucose** concentration of 200 mg/100 ml the sensitivity is 98% and the specificity 99%. As an investigator can perform per day 300 double determinations and as the expenses for the reagents per test amount to only 0.005 marks, this method appears to be suited for a diabetes screening by way of a postprandial **glucose** determination. For this purpose, furthermore, a simple method of processing capillary **blood** is proposed for the **glucose** determination that avoids deproteinization and centrifugation.

L14 ANSWER 43 OF 43 MEDLINE
AN 71210523 MEDLINE
DN 71210523
TI **Hexokinase**: a **compartmented** enzyme.
AU Anderson J W; Herman R H; Tyrrell J B; Cohn R M
SO AMERICAN JOURNAL OF CLINICAL NUTRITION, (1971 Jun) 24 (6) 642-50.
Ref: 80
Journal code: 3EY. ISSN: 0002-9165.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197109

=> d his l15-; d 1-14 bib abs

(FILE 'CAPLUS, BIOSIS, MEDLINE, EMBASE, LIFESCI, BIOTECHDS, WPIDS, CONFSCI, DISSABS, SCISEARCH, JICST-EPLUS, PROMT, USPATFULL' ENTERED AT 15:49:53 ON 26 OCT 1998)

Author(s)

L15 10946 S WONG S?/AU
L16 47 S HILTIBRAN R?/AU
L17 8732 S HUANG T?/AU
L18 0 S L15 AND L16 AND L17
L19 12 S L15 AND (L16 OR L17)
L20 0 S L16 AND L17
L21 17 S (L15 OR L16 OR L17) AND L3
L22 29 S L19 OR L21
L23 14 DUP REM L22 (15 DUPLICATES REMOVED)

L23 ANSWER 1 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 1
AN 1998:271121 CAPLUS
DN 129:26277

TI Neuronal overexpression of heme oxygenase-1 correlates with an attenuated exploratory behavior and causes an increase in neuronal NADPH **diaphorase** staining

AU Maines, M. D.; Polevoda, B.; Coban, T.; Johnson, K.; Stoliar, S.; **Huang, T. J.**; Panahian, N.; Cory-Slechta, D. A.; WcCoubrey, W. K., Jr.

CS Departments of Biochemistry and Biophysics, Environmental Medicine, and Neurobiology/Anatomy, University of Rochester School of Medicine, Rochester, NY, USA

SO J. Neurochem. (1998), 70(5), 2057-2069
CODEN: JONRA9; ISSN: 0022-3042

PB Lippincott-Raven Publishers

DT Journal

LA English

AB Heme oxygenase isoenzymes, HO-1 (also known as hsp32) and HO-2, are the source for the formation of the putative messenger mol. carbon monoxide (CO), reactive iron, and the in vitro antioxidant bilirubin. We have developed and characterized transgenic (Tg) mice that overexpress the stress protein in neurons in various brain regions. The Tg mice were generated by the use of rat HO-1 cDNA under the control of the neuron-specific enolase promoter. Except for a tendency to have an enlarged spleen, Tg mice did not show gross anatomical changes. Increase in HO-1 mRNA, which was demonstrated by northern blot anal. and in situ hybridization, was accompanied by an increase in neuronal HO-1 protein expression, shown by immunohistochem. and western blotting, and an increase in HO activity. Expression of the transgene correlated with an attenuation of exploratory behavior and increased circling activity and coincided with enhanced neuronal NADPH **diaphorase** staining. Those changes were not accompanied by an increase in DNA damage or significant change in whole-brain NO synthase activity. The HO-1 Tg mice potentially represent a good model to examine the function of CO as a neuromodulator, iron as a gene regulator, and bile pigments as in vivo antioxidants.

L23 ANSWER 2 OF 14 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.DUPLICATE 2

AN 97301519 EMBASE

TI Grafts of EGF-responsive neural stem cells derived from GFAP-hNGF transgenic mice: Trophic and tropic effects in a rodent model of huntington's disease.

AU Kordower J.H.; Chen E.-Y.; Winkler C.; Fricker R.; Charles V.; Messing A.; Mufson E.J.; **Wong S.C.**; Rosenstein J.M.; Bjorklund A.; Emerich D.F.; Hammang J.; Carpenter M.K.

CS Dr. J.H. Kordower, Department of Neurological Sciences, Rush Presbyterian Medical Center, 2242 West Harrison Street, Chicago, IL 60612, United States. jkordowe@rush.rpslmc.edu

SO Journal of Comparative Neurology, (1997) 387/1 (96-113).
Refs: 47
ISSN: 0021-9967 CODEN: JCNEAM

CY United States
DT Journal
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LA English
SL English
AB The present study examined whether implants of epidermal growth factor (EGF)responsive stems cells derived from transgenic mice in which the glial fibrillary acid protein (GFAP) promoter directs the expression of human nerve growth factor (hNGF) could prevent the degeneration of striatal neurons in a rodent model of Huntington's disease (HD). Rats received intrastriatal transplants of GFAP-hNGF stem cells or control stem cells followed 9 days later by an intrastriatal injection of quinolinic acid (QA). Nissl stains revealed large striatal lesions in rats receiving control grafts, which, on average, encompassed 12.78 mm3. The size of the lesion was significantly reduced (1.92 mm3) in rats receiving lesions and GFAP-hNGF transplants. Rats receiving QA lesions and GFAP-hNGF-secreting grafts stem cell grafts displayed a sparing of striatal neurons immunoreactive (ir) for glutamic acid decarboxylase, choline acetyltransferase, and neurons histochemically positive for nicotinamide adenosine diphosphate. Intrastriatal GFAP-hNGF- secreting implants also induced a robust sprouting of cholinergic fibers from subjacent basal forebrain neurons. The lesioned striatum in control-grafted animals displayed numerous p75 neurotrophin-ir (p75(NTR)) astrocytes, which enveloped host vasculature. In rats receiving GFAP-hNGF-secreting stem cell grafts, the astroglial staining pattern was absent. By using a mouse-specific probe, stem cells were identified in all animals. These data indicate that cellular delivery of hNGF by genetic modification of stem cells can prevent the degeneration of vulnerable striatal neural populations, including those destined to die in a rodent model of HD, and supports the emerging concept that this technology may be a valuable therapeutic strategy for patients suffering from this disease.

L23 ANSWER 3 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS
AN 91:377686 BIOSIS
DN BR41:50076
TI EFFECT OF ALUMINUM ON BRAIN GLUCOSE METABOLISM.
AU MARCUS D L; **WONG S**; FREEDMAN M L
CS DEP. MED., DIV. GERIATRICS, NYU MED. CENT., NEW YORK, NY.
SO JOINT MEETING OF THE ASSOCIATION OF AMERICAN PHYSICIANS, THE AMERICAN SOCIETY FOR CLINICAL INVESTIGATION, AND THE AMERICAN FEDERATION FOR CLINICAL RESEARCH, SEATTLE, WASHINGTON, USA, MAY 3-6, 1991. CLIN RES 39 (2). 1991. 448A. CODEN: CLREAS ISSN: 0009-9279
DT Conference
LA English

L23 ANSWER 4 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 3
AN 1990:500708 CAPLUS
DN 113:100708
TI Lubricant production process
IN Chen, Nai Y.; Garwood, William E.; **Huang, Tracy J.**; Le, Quang N.; **Wong, Stephen S.**
PA Mobil Oil Corp., USA
SO U.S., 22 pp. Cont.-in-part of U.S. Ser. No. 793,937, abandoned. CODEN: USXXAM
PI US 4919788 A 19900424
AI US 88-260857 19881021
PRAI US 84-685089 19841221
US 85-793937 19851101
US 86-821125 19860122
DT Patent
LA English
AB Lubricating oils of low pour point and high viscosity index are produced by 1st carrying out a partial dewaxing of a lubricant base stock in an initial catalytic operation, followed by a selective dewaxing step. The initial dewaxing step is carried out using a large-pore, high silica zeolite dewaxing catalyst such as zeolite Y or beta which dewaxes by isomerizing the waxy components of the base stock to less waxy branched-chain isoparaffins but the severity of the operation is controlled so as to effect only a partial removal of the waxy components. Further removal of the waxy components is effected during the subsequent dewaxing step which is selective for the removal of the more waxy n-paraffin components, leaving the branched-chain isoparaffins which contribute to a high-viscosity index in the product. The selective dewaxing step may be a solvent, e.g., MEK dewaxing operation or a catalytic dewaxing using a highly shape-selective zeolite such as ZSM22 or ZSM23. The pour point of the feedstock is preferably reduced during the initial catalytic dewaxing to a value which is >10.degree.F and preferably >20.degree.F, above the target pour point for the products. Generally, this will entail a redn. of >10.degree.F and preferably >20.degree.F in the pour point of the feed.

L23 ANSWER 5 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 4
AN 1987:410262 CAPLUS
DN 107:10262
TI Redispersion of agglomerated noble metals on zeolite catalysts
IN Borghard, William S.; **Huang, Tracy J.**; McCullen, Sharon B.; Schoennagel, Hans J.; Tsao, Ying Yen P.; **Wong, Stephen S.**
PA Mobil Oil Corp., USA
SO U.S., 6 pp. Cont.-in-part of U.S. Ser. No. 778,299 abandoned. CODEN: USXXAM
PI US 4657874 A 19870414
AI US 86-819074 19860115

PRAI US 84-571760 19840118
US 85-710515 19850311
US 85-778299 19850923
DT Patent
LA English
AB A process for regeneration of a deactivated noble metal-contg. zeolite catalyst (esp. from reforming of hydrocarbons) comprises redispersing the noble metals assocd. with the deactivated catalyst (contg. SiO2-Al2O3 ratio .gtoreq.20:1) by (a) contacting the catalyst with a stream of inert gas contg. Cl, water, and optionally with O at 150-450.degree., 6-15 Torr partial pressure Cl, and a ratio of partial pressure of water to partial pressure of Cl of 0.01-2:1; (b) purging the resulting catalyst with an inert gas, and (c) reducing the purged catalyst with H at 140-550.degree.. Thus, a 0.6 wt.% Pt-zeolite beta catalyst was regenerated by contacting with a stream of N contg. water, Cl, and O at partial pressures of 14, 12, and 40 Torr, resp., for 4 h; after purging with N, the catalyst was reduced with H at 450.degree. for 1 h, resulting in increase of the Pt dispersion in the catalyst from 6% to 28%.

L23 ANSWER 6 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 5
AN 1987:220986 CAPLUS
DN 106:220986
TI Regeneration of noble metal-highly siliceous zeolite catalysts with sequential hydrogen halide and halogen or organic halogen compound treatment
IN McCullen, Sharon B.; **Wong, Stephen S.**; **Huang, Tracy J.**
PA Mobil Oil Corp., USA
SO U.S., 5 pp. Cont. of U.S. Ser. No. 580,925, abandoned.
CODEN: USXXAM
PI US 4645751 A 19870224
AI US 85-814082 19851223
PRAI US 84-580925 19840216
DT Patent
LA English
AB A process for rejuvenation of a deactivated, highly siliceous noble metal-contg. zeolite catalyst (e.g., Pt-zeolite beta) which contains agglomerated noble metals comprises initially reducing the deactivated catalyst in H, pretreating the reduced catalyst with an inert gas stream contg. .apprx.0.001-10 wt.% of a halide (e.g., HCl), redispersing the noble metal with an inert gas stream contg. .apprx.0.001-10 wt.% of a halogen or halogen-contg. org. material (e.g., Cl2 or CH2Cl2), and subsequently reducing the catalyst. The halogen treatments may optionally occur in the presence of O or sources of O. The process is applicable to ZSM zeolites, zeolite Y, and zeolite beta.

L23 ANSWER 7 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 6

AN 1987:531294 CAPLUS
DN 107:131294
TI Enzymic differences between hycanthone-resistant and sensitive strains of Schistosoma mansoni
AU Doong, Yung Chuen; Wong, Lee Jun C.; Bruce, John I.; **Wong, Shan S.**
CS Dep. Chem., Univ. Lowell, Lowell, MA, 01854, USA
SO Comp. Biochem. Physiol., B: Comp. Biochem. (1987), 87B(3), 459-64
CODEN: CBPBB8; ISSN: 0305-0491
DT Journal
LA English
AB Hycanthone (I)-sensitive and -resistant adult worms of S. mansoni had generally similar specific activities in 10 enzymes of carbohydrate metab. Kinetic analyses revealed that pyruvate kinase, glucose 6-phosphate (G6P) dehydrogenase, and malate dehydrogenase from both strains possessed similar Michaelis-Menten consts. and were not inhibited by I. **Hexokinase** and lactate dehydrogenase from the drug-resistant strain were not inhibited by I and showed 3-5-fold greater Km values than did those from the drug-sensitive worms which were also inhibitable by I. I more drastically affected the Vmax of phosphofructokinase from the I-sensitive parasite. Hence, the I inhibitable enzymes are generally from the drug-sensitive strain, whereas the enzymes from drug-resistant worms are mostly I insensitive.

L23 ANSWER 8 OF 14 LIFESCI COPYRIGHT 1998 CSA
AN 87:84572 LIFESCI
TI Enzymatic differences between hycanthone-resistant and sensitive strains of Schistosoma mansoni .
AU Doong, Yung-Chuen; Wong, Lee-Jun C.; Bruce, J.I.; **Wong, Shan S.**
CS Biochem. Program, Dep. Chem., Univ. Lowell, Lowell, MA 01854, USA
SO COMP. BIOCHEM. PHYSIOL., B., (1987) vol. 87B, no. 2, pp. 459-464.
DT Journal
FS L
LA English
SL English
AB Hycanthone-sensitive and resistant adult worms of Schistosoma mansoni were found to have generally similar specific activities in ten enzymes of carbohydrate metabolism. Kinetic analyses revealed that pyruvate kinase, glucose-6-phosphate (G6P) dehydrogenase and malate dehydrogenase from both strains possessed similar Michaelis-Menten constants and were not inhibited hycanthone. **Hexokinase** and lactate dehydrogenase from the drug-resistant strain were not inhibited by hycanthone and showed three to five times greater K sub(m) values than those from the drug-sensitive worms which were also inhibitable by hycanthone.

L23 ANSWER 9 OF 14 CAPLUS COPYRIGHT 1998 ACS

AN 1987:425269 CAPLUS
DN 107:25269
TI A group contribution theory for modeling phase equilibria of carbon dioxide and hydrocarbons
AU **Huang, Tze Jyh; Wong, Shan Hill**
CS Dep. Chem. Eng., Natl. Tsing Hua Univ., Hsinchu, 300, Taiwan
SO J. Chin. Inst. Chem. Eng. (1987), 18(1), 53-5
CODEN: JCICAP; ISSN: 0368-1653
DT Journal
LA English
AB A cubic equation of state was used to model the phase equil. in supercrit. fluid extn. The binary interaction coeff. used in the equation of state was estd. by using the excess Gibb's free energy at the limit of infinite pressure. This Gibb's free energy was estd. by the UNIFAC group contribution method. The resulting model correlates exptl. data for CO2 and naphthenic hydrocarbons.

L23 ANSWER 10 OF 14 USPATFULL
AN 85:10396 USPATFULL
TI Conversion of Fischer-Tropsch products
IN Chen, Nai Y., Titusville, NJ, United States
Haag, Werner O., Lawrenceville, NJ, United States
Huang, Tracy J., Lawrenceville, NJ, United States
Wong, Stephen S., Langhorne, PA, United States
PA Mobil Oil Corporation, New York, NY, United States (U.S. corporation)
PI US 4500417 850219
AI US 82-453970 821228 (6)
DT Utility
EXNAM Primary Examiner: Gantz, Delbert E.; Assistant Examiner: Chaudhuri, O.
LREP McKillop, Alexander J.; Gilman, Michael G.; Santini, Dennis P.
CLMN Number of Claims: 16
ECL Exemplary Claim: 1,12
DRWN No Drawings
LN.CNT 676
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The high boiling fraction of Fischer-Tropsch synthesis is upgraded by contact with a catalyst comprising a high-silica, large pore zeolite and a hydrogenation component. The products are a distillate fraction characterized by low sulfur and nitrogen content, high isoparaffin content and low pour point and a lube fraction characterized by its high viscosity index and low pour point.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L23 ANSWER 11 OF 14 WPIDS
AN 84-207236 [34] WPIDS

DNC C84-087192
TI Upgrading high-boiling Fischer-Tropsch synthesis product fractions - by conversion over zeolite-contg. catalyst.
DC H04 J04
IN CHEN, N Y; HAAG, W O; **HUANG, T J H; WONG, S S F**
PA (MOBI) MOBIL OIL CORP
CYC 4
PI AU 8321809 A 840705 (8434)* 25 pp
JP 59133290 A 840731 (8436)
US 4500417 A 850219 (8510)
ZA 8309664 A 850628 (8539)
ADT AU 8321809 A AU 83-21809 831129; JP 59133290 A JP 83-244988 831227; US 4500417 A US 82-453970 821228; ZA 8309664 A ZA 83-9664 831228
PRAI US 82-453970 821228
AN 84-207236 [34] WPIDS
AB AU 8321809 A UPAB: 930925
Upgrading of high-boiling fractions of Fischer-Tropsch synthesis prods. is effected by contact at 200-500 deg.C, 500-20000 kPa and an LHSV of 0.1-20 with a catalyst comprising (a) a hydrogenation component (I) and (b) a large-pore crystalline zeolite (II) having a SiO2/Al2O3 ratio of at least 10:1. The pref. (I) are combinations of Gp.VA, VIA, VIIA, VIIIA, IB, IIB or IVB, e.g. Co/Mo, Ni/W, Pt/Ir, Pt/Re and Pt/Ir/Re.

USE/ADVANTAGE - Fischer-Tropsch fractions having an i.b.p. of at least 150 (pref. at least 174, esp. at least 343) deg.C are upgraded to distillate boiling range prods. having pour points of below -18 deg.C and high VI lubricants having pour points of below 0 deg.C.

0/0

ABEQ US 4500417 A UPAB: 930925
Heavy oil fraction of initial b.pt. 125 deg.C or more produced in Fischer-Tropsch syntheses, is upgraded by contacting fraction and H2 at 200-500 deg.C, H2-partial pressure of 500-20,000 kPa and liq. hourly space velocity of 0.1-20, with a zeolite beta catalyst composited with a hydrogenation metal to form a hydrocarbon distillate fraction and paraffin-type heavy hydrocarbon fraction for conversion to lubricant oil.

Pref. silica-alumina ratio of zeolite is 50:1 (pref. 100:1). Hydrogenation cpd. is based on Gp. VA, VIA, VIIA, VIIIA, IB, IIB and/or IVB metal(s). Heavy oil fraction has b.pt. more than 174 deg.C (pref. 3 x 3 deg.C).

ADVANTAGE - Prods. comprise a low S and N-contg. distillate fraction of high isoparaffin content and low pour point; and a lute fraction of high viscosity index and low pour point.

L23 ANSWER 12 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS
AN 82:78008 BIOSIS
DN BR23:8000
TI 1 STEP DETERMINATION OF HYDROGEN TRANSFER STEREOSPECIFICITY OF

NICOTINAMIDE NUCLEOTIDE LINKED OXIDO REDUCTASES.
AU **WONG S S**
CS DEP. CHEM., UNIV. LOWELL, LOWELL, MASS. 01854.
SO 26TH ANNUAL MEETING OF THE BIOPHYSICAL SOCIETY, BOSTON, MASS., USA, FEB. 14-17, 1982. BIOPHYS J 37 (2 PART 2). 1982. 74A. CODEN: BIOJAU ISSN: 0006-3495
DT Conference
LA English

L23 ANSWER 13 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 7
AN 1981:438017 CAPLUS
DN 95:38017
TI NMR determination of hydrogen transfer stereospecificity of .alpha.-keto acid dehydrogenase complexes: a one-step method
AU Zhou, Meng-Ai; **Wong, Shan S.**
CS Dep. Chem., Univ. Lowell, Lowell, MA, 01854, USA
SO J. Biochem. Biophys. Methods (1981), 4(5-6), 329-38 CODEN: JBBMDG; ISSN: 0165-022X
DT Journal
LA English
AB A simple 1-step method is described for the detn. of the H transfer stereospecificity of NAD-linked oxidoreductases. Alc. and lipoamide dehydrogenases, whose stereospecificities are known, are employed to prep. stereospecifically deuterated NADH which is immediately reoxidized in situ in a NMR tube by the enzyme under investigation. Alternatively, NADH produced by the test enzyme is reoxidized in situ by enzymes of known stereospecificity, such as glutamate and lactate dehydrogenases. The reoxidized coenzyme of the coupled reactions is analyzed for its 2H content by NMR spectroscopy. The presence and absence of the absorption band due to 1H or 2H, resp., at the 4-position of the nicotinamide ring is used to diagnose the stereospecificity of the test enzyme. Application of this direct in situ coupling method to dihydrolipoyl dehydrogenases of Escherichia coli pyruvate and .alpha.-ketoglutarate dehydrogenase complexes indicates that the enzymes are B-side stereospecific for NAD.

L23 ANSWER 14 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS
AN 79:157177 BIOSIS
DN BA67:37177
TI ESCHERICHIA-COLI PYRUVATE DEHYDROGENASE COMPLEX SITE COUPLING IN ELECTRON AND ACETYL GROUP TRANSFER PATHWAYS.
AU FREY P A; IKEDA B H; GAVINO G R; SPECKHARD D C; **WONG S S**
CS DEP. CHEM., OHIO STATE UNIV., COLUMBUS, OHIO 43210, USA.
SO J BIOL CHEM 253 (20). 1978 7234-7241. CODEN: JBCHA3 ISSN: 0021-9258
LA English
AB The following experiments showed that half the .alpha.-lipoyl moieties in the E. coli pyruvate dehydrogenase complex are coupled to the dihydrolipoyl dehydrogenase (E3.cntdot.FAD) component of the complex and to DPNH formation. Reductive acetylation of the complex

by [2-14C]pyruvate in the absence of DPN+ and CoA produces a [1-14C]acetylpyruvate dehydrogenase complex. Up to 10.7 nmol of [1-14C]acetyl groups/mg of complex can be incorporated in this way. The reaction of the acetylated complex with CoA and DPN+ produces a maximum of only 5.2 nmol of DPNH/mg of complex, just half the number of acetyl groups and electron pairs introduced from pyruvate. Once the complex is permitted to catalyze the conversion of pyruvate to acetylCoA, DPNH, and CO2 through one catalytic turnover, reductive acetylation with [2-14C]pyruvate introduces only 5.3 nmol of [1-14C]acetyl groups/mg of complex. Only half the sites could be acetylated because in the first turnover only half of the dihydrolipoyl moieties were oxidized by DPN+. The complex can be acetylated by [3H]CoA in a DPNH-dependent reaction. The maximum number of [3H]acetyl groups that can be incorporated in this way corresponds to 5.5 nmol/mg of protein. Only half the .alpha.-lipoyl groups could be reductively acetylated by acetylcoenzyme A and DPNH because only half could be reduced by DPNH. It is concluded that there are 2 classes of .alpha.-lipoyl groups associated with dihydrolipoyl transacetylase (E2), the core enzyme of this complex. One class consists of 5.2-5.5 nmol/mg of complex that are coupled to both the pyruvate dehydrogenase (E1) and the dihydrolipoyl dehydrogenase (E3.cntdot.FAD) components. This class is involved in catalyzing the reaction pyruvate + CoA + DPN+ .fwdarw. CO2 + acetylCoA + DPNH. The 2nd class consists of an equal number of .alpha.-lipoyl groups on the core enzyme that are coupled to the pyruvate dehydrogenase (E1) component but not to dihydrolipoyl dehydrogenase (E3.cntdot.FAD). This latter class has another so far undefined electron transfer function in the E. coli cell.

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FILE 'HOME' ENTERED AT 15:53:17 ON 26 OCT 1998